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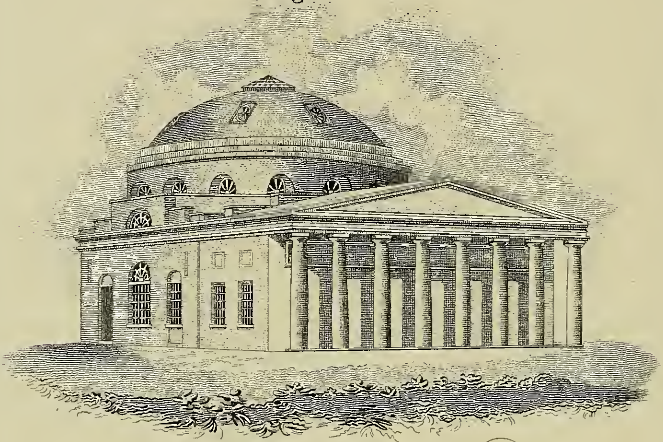

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EXPERIMENTAL RENAL HYPERTENSION*†

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Certain new technics developed in the last decade give promise of solving the problem of the origin of essential hypertension. This disease, a primary contributing cause to more deaths than cancer, kills over 375,000 individuals annually in the United States alone. Physiologists have long been aware that persistently high blood pressures are most commonly caused either by increased cardiac output or increased peripheral resistance. In clinical hypertension the latter is almost certainly the cause, but the reason why in certain individuals the arterioles should decrease their diameters and so obstruct the flow of blood could only be guessed at until recently. If the understanding of this problem can be bettered by any new approach it is a matter of importance to the medical profession. Such new approaches are now available. It is the purpose of this discussion to survey them briefly and in doing so to consider the problem of experimental renal hypertension, to discuss the manner in which it may be produced, what the mechanisms of its origins are, and what bearing it has on the clinical aspects of hypertension. For detailed treatment of this subject the interested reader is referred to two excellent reviews, one by Blalock (2) and the other by Page (26).

PRODUCTION OF EXPERIMENTAL RENAL HYPERTENSION

A relationship between kidney function and high blood pressure has long been known clinically. Experimentally, hypertension may be produced by kidney injury but until recently such injuries usually resulted in the death

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of the animal within a few days. In particular, interference with the circulation of the kidney has been recognized for some time to produce hypertension, but the problem has been how to accomplish this and still keep the animal alive and reasonably healthy for long enough periods to enable the proper sort of scientific investigation to be made. Methods for doing this are now at hand.

In 1933, Loesch (22) working in this country exteriorized the kidney of a dog in such a manner that its circulation, arterial and venous, could be completely shut off by clamping. This was done at intervals of every two or three days for fifteen to thirty minutes at a time. Shortly after initiating such procedures the blood pressure slowly began to rise and continued to do so for some weeks until it attained a permanent level of between 170 and 200 mm. hg. Here it stayed as long as the experiment was continued, which in several instances was for two or three months.

The now widely known and almost classical experiments of Goldblatt (9) were published in 1934. With the use of dogs he devised a silver clamp which could be placed about the renal artery and left in position indefinitely. When the clamp was closed to partly occlude the arterial circulation a gradual rise in blood pressure took place over a period of several weeks. As long as the clamp was not disturbed the pressure continued to rise until it reached the level of about 200 mm. hg., where it remained until the clamp was removed. Many of Goldblatt's dogs survived such treatment for several years. The severity of the hypertension was correlated with the degree of clamping. In those animals with almost completely occluded arteries, kidney dysfunction occurred and the animal died. With less severe clamping no obvious signs of kidney damage could be seen. Complete closure of the artery produced no rise in blood pressure. Either one or both of the kidneys may be involved.

The Goldblatt method, with modifications, has been adopted by many investigators and has proved the most reliable for work with large animals. Numerous studies have been made on experimental hypertension produced in this way and the results published. The literature is already extensive. These studies may ultimately solve the problem of the origin of benign and malignant hypertension. Blalock in his recent review of experimental hypertension states that "while there are some types of hypertension that are non-renal in origin the evidence . . . indicates that most instances of experimental and probably clinical hypertension are related to some abnormality in function of the kidneys."

Other methods for producing experimental hypertension have been developed. One has been worked out by Chanutin and Ferris (5) which has proved useful in working with small animals where the Goldblatt procedure is impractical. The removal of one-half to two-thirds of each kidney from the circulation is accomplished either by ligation or excision by a two stage operation. In recovery there is a gradual development over a period of two or three months of hypertension. This is accompanied by polyuria, and Chanutin and Ferris advance the interesting suggestion that possibly the rise in blood pressure is a compensatory mechanism designed to produce a sufficiently diluted urine to remove the metabolites in low concentration.

Hypertension can also be produced by ligating one ureter, the resulting hydronephrosis probably producing an ischemia in the affected kidney. Complete occlusion of the renal artery in a unilateral nephrectomized cat for four or five hours will on release of the clamp likewise produce an immediate rise in blood pressure. This type of hypertension resembles those produced by other methods and is useful in acute experiments when more extensive surgical intervention is to be avoided.

PROPERTIES OF RENIN

The blood pressure rises following these procedures because the ischemic kidney discharges into the circulation increased amounts of a pressor substance known as renin. This is the generally accepted explanation. As far back as 1898 Tigerstedt and Bergmann (31) extracted a pressor substance from the kidney of a normal rabbit which when injected into another rabbit produced a sustained rise in blood pressure. The substance was called renin. Their conclusions with regard to its nature are substantially those that prevail today, with one exception. This is the belief that renin acted not directly on the arteriolar musculature but indirectly through the peripheral autonomic ganglia. In all else they have been corroborated fully by later work. They showed it to be effective in pithed preparations and so eliminated the possibility of reflex mediation through the central nervous system. Their renin was also destroyed by heating, was dialyzable, and soluble in water and alcohol. The work of Tigerstedt and Bergmann did not attract much attention at the time of its publication and soon lapsed into semiobscurity. Interest in it, however, enjoyed a sharp revival when it was seen to bear directly upon the recent studies on experimental hypertension.

The early work on renin was subject to criticism on the ground that the extracts also contained a depressor substance and the first effect of an injection was to lower the blood pressure. This objection has been overcome and today methods are available for the preparation of renins which do not give any preliminary fall in blood pressure [Pickering and Prinzmetal (27), Grossman (12), and Helmer and Page (15)]. Investigators agree that the best source of renin is the cortex of the kidney rather than the medulla. It has been amply demonstrated that renin is not to be confused with adrenalin, pituitrin, tyramine or any similar pressor substance which can be extracted from other organs and tissues. Leo, Prinzmetal, and Lewis (20) have shown that its action persists after the injection of drugs which reverse the effect of adrenalin in raising the blood pressure.

THE ISCHEMIC KIDNEY

Tigerstedt and Bergmann (31) worked with healthy rabbits and extracted their renin from normal kidneys. Is renin also present in ischemic kidneys and, if so, in greater or less amounts? If the hypertension of renal ischemia is caused by renin, then presumably more of this substance is discharged into the circulation from an ischemic kidney than a normal one. Actually there is considerably more renin in an ischemic kidney than in a normal one. This has been amply demonstrated by numerous investigators, among them Harrison, Blalock, Mason, and Williams (13), and Pickering and Prinzmetal (27).

The reason why an ischemic kidney puts out more renin than a normal one has naturally attracted a great deal of attention. If renin is implicated in clinical hypertension, obviously the answer to this becomes of fundamental importance. Numerous studies have been made on blood flows, oxygen consumptions and clearances of ischemic kidneys without any decisive results. Goldblatt, Lynch, Hanzal, and Summerville (9) showed that hypertension could be produced in an animal by clamping on the renal artery so slightly as not to produce any disturbance in kidney function as judged by the urea clearance test. Grossly, the ischemic kidneys were somewhat reduced in size, as might be expected; microscopically, there was thickening of the Bowman's capsule and of the basement membranes of the glomerular tufts. Levy, Light, and Blalock (21) found the reduced blood flow through an ischemic kidney was not accompanied by any change in the arteriovenous oxygen difference. This would seem to indicate either a lowered metabolism of the ischemic kidney or a destruction of kidney tissue. Kohlstaedt and Page (18) suggested that the increased production of renin resulted from a reduction in pulse pressure in the ischemic kidney rather than a reduction of blood pressure or blood flow. Dock and Rytand (7) found the blood flow to be the same through the remaining kidney after unilateral nephrectomy as through a kidney subjected to subtotal nephrectomy, although hypertension developed only in the latter case. All of this is confusing and admittedly subject to no simple interpretation. More work is needed on this aspect of the problem.

TACHYPHYLAXIS

One of the puzzling features of the action of renin when injected is the development of so-called tachyphylaxis or tolerance. This was originally described by Tigerstedt and Bergmann (31). As the initial dose is repeated the pressor response becomes less each time until it disappears entirely. According to Taggart and Drury (30), it hardly seems reasonable to believe that renin could be responsible for renal hypertension if tolerance to it is developed so quickly. Page (25) believes that tachyphylaxis results from the exhaustion of a renin activator in the blood. The evidence for this will be considered in greater detail later. Leo, Prinzmetal, and Lewis (20) observed that hypertension following the termination of complete renal ischemia in the cat did not occur if the animal was previously rendered tachyphylactic to renin. This they took as evidence that renin was implicated in the hypertension usually developed under these circumstances.

DEMONSTRATION OF PRESSOR SUBSTANCE IN BLOOD

Investigators have been searching for years for a pressor substance in the blood of individuals suffering from essential hypertension. As is well known, this search has been fruitless. Yet if the current theory of renal hypertension is correct such a substance should be found or else an adequate explanation devised for its absence. If such a pressor substance is to be found anywhere it should be in blood drawn from animals with ischemic kidneys, if not in the general circulation then certainly in the venous blood as it flows from the kidney. Tigerstedt and Bergmann made the original attempt in 1898 and thought they were successful, although the evidence was not conclusive. However, Braun-Menendez, Fasciolo, Leloir, and Muñoz (4) were able to make a convincing demonstration. They did not consider their substance to be renin but something resulting from the action of renin on a pseudoglobulin substrate to produce what they called hypertensin. It was dialyzable through cellophane while renin, of course, is not. Friedman, Selzer, and Sampson (8) also found a pressor substance in the venous blood of ischemic kidneys. This they felt was not renin nor did they believe it to be hypertensin since it could be dialyzed through cellophane. Its concentration in the venous blood was as high fifteen minutes after clamping as it was several hours later. Beckwith (1) has reported that the amount of pressor substance extractable from an ischemic kidney is at its greatest one-half hour after complete ligation of the artery and from then on progressively declines, until after sixteen hours the amount found is less than that in a normal kidney. Apparently an enzyme substance in the completely ischemic kidney destroys the pressor substance rather quickly. The rapid rise in the concentration of pressor substance in these kidneys is in sharp contrast to the slowly developing hypertension of the Goldblatt preparation.

Certain relationships between the pressor substance of the kidneys and other glands of internal secretion are worth noting here. Ogden, Brown, and Page (24) observed a marked increase in the sensitivity of the arterioles to pitressin after partial occlusion of the renal artery. This effect appears before there is a marked elevation in blood pressure. If pitressin does have any influence on arterial tone this fact has interesting implications. Blalock and Levy (3), confirming Goldblatt, detected a disappearance of experimental renal hypertension after bilateral adrenalectomy. The effect seemed to develop in a day or so, much sooner than an adrenal cortical deficiency could possibly occur. Injections of cortical hormone were unable to restore the blood pressure to its former hypertensive level, nor was sodium chloride therapy effective. Remington, Collings, Hays, Parkins, and Swingle (28) found that the pressor response to small doses of renin was less than usual in an adrenalectomized animal. This was also accompanied by a loss of sensitivity to pitressin and barium chloride. Swingle is therefore inclined to attribute the decrease in renin response to a general loss of vascular tone resulting from the absence of the cortical hormone.

ANGIOTONIN

That renin is not the actual pressor substance directly responsible for hypertension but only a precursor thereto has been suggested by several investigators. As the purification of renin proceeds it becomes progressively less and less active and dialyzed renin is totally inactive (17). If this inactive preparation is mixed with dog's blood (also inactive) and added to a perfusate flowing through the isolated tail of a dog, a distinct vasoconstriction occurs. Page (25) later extended this work by using purified renin. A rabbit's ear was perfused with dog's blood. Each time renin was added to the perfusate there was a gradual diminution in the vasoconstriction observed until finally the addition of further renin had no effect. If at this point an "activator" derived from oxblood serum was added to the perfusate there was an immediate vasoconstriction. A Ringer-Locke-acacia solution, plus renin, used as a perfusate in the same type of preparation produced no reduction in the blood flow, but if to this solution an activator was also added vasoconstriction did occur. Later Kohlstaedt, Page and Helmer (19) demonstrated an activator in both red cells and plasma and showed that the blood of the dog in the Goldblatt experiment contained more activator than that of a normal dog.

Finally Helmer and Page (15), on mixing relatively pure preparations of renin and renin-activator (derived from oxblood by fractional precipitation with potassium phosphate), isolated a crystalline substance having appreciable pressor effects. This they called angiotonin and thought it the active agent responsible for the hypertension of experimental renal ischemia. The renin released from the ischemic kidneys combined in the blood with the activator to form angiotonin. This in turn acted directly on the blood vessel wall and produced contraction. Renin was thought to be an enzyme acting on the activator substrate. Tachyphylaxis to angiotonin is produced more slowly than to renin, but it does occur. For this reason Page (26) believes there must also be an activator in the blood for angiotonin. Just what the relationship between angiotonin, hypertensin and the pressor substance of Friedman, Selzer and Sampson (8) is, remains to be seen.

Corcoran and Page (6) found a reduced inulin clearance in the normal kidney on injection of angiotonin. At the same time, the inulin extraction percentage rose and the plasma renal flow decreased. This was interpreted as indicating a constriction of the efferent glomerular artery, an orthodox deduction in accord with modern theories as to renal function. The constricted efferent artery so reduced flow through the glomerulus that there was more time available for filtration, thus raising the inulin extraction percentage. Corcoran and Page point out that changes similar to these are reported in arterial hypertension in man by Smith, Goldring, Chasis, and Ranges (29).

EVIDENCE FOR AN ANTIPRESSOR SUBSTANCE IN KIDNEY

The existence of renin in the normal kidney could mean that it has some physiologic importance. Conceivably it might act to aid in the maintenance of normal tone in the vascular wall, a function which has been assigned by others on occasion to pitressin and cortin. Such speculation is idle, however, until more is known about the physiology of renin. As yet no one has attempted a continuous experiment designed to reveal the effects of a complete absence of renin.

However, assuming that renin does have a rôle in normal physiology, an antagonist to it might exist. The presence of an antipressor substance in the kidney has already been suggested by earlier work. Tigerstedt and Bergmann (31) found nephrectomized rabbits to be much more sensitive to renin than normal ones. Helmer and Page (15) similarly observed smaller rises in blood pressures in normal animals than in nephrectomized ones. On this basis they postulated the presence of an inhibitor in the normal kidney which acted to reduce the pressor effects of renin. Because of the possible clinical importance of such an inhibitor several independent groups have been studying it. Jablons (16) announced the isolation of an extract called tubulin from the kidneys of hogs. It was protein and lipoid free, and soluble in water and alcohol. Tubulin when administered to hypertensive patients often reduced their blood pressures. Jablons reported upon the results of several cases, about half of whom showed improvement with this extract.

The greater sensitivity of nephrectomized animals to renin might be attributable not to the absence of an antagonist but to a failure to excrete renin. Merrill, Williams and Harrison (23) concluded this was not so, as they found no increase in sensitivity to renin until two days after nephrectomy. Harrison, Grollman and Williams (14) then proceeded to search for an antipressor substance in the kidney. They prepared an extract of pig's kidneys according to the method described by Grollman, Williams and Harrison (10), which was water-soluble but not soluble in alcohol or other organic solvents and was nonprotein in nature. Such an extract acted to lessen the pressor effect of injected renin, although it did not abolish it completely. On normal rats the extract had no effect whatever on blood pressure. In rats made hypertensive by subtotal nephrectomy the extract brought the blood pressure back to normal levels. Highly important is the fact that it was effective by mouth, thus sharply differentiating it from any other depressor extract such as histamine.

In the course of these experiments hypertensive pregnant rats were noted to show a gradual reduction in blood pressure as they approached term. This the observers attributed to the presence of antipressor substance in the maternal circulation derived from the fetal kidney. Following parturition the blood pressure returned to hypertensive levels. Similarly, renin was less effective in hypertensive rats. Such experiments as these are highly suggestive and support the claim than an antipressor substance is present in the kidney.

Grollman, Williams and Harrison (11) reported on the clinical use of their extract in various types of hypertension. In most cases moderate declines in both systolic and diastolic blood pressures occurred on oral administration. The results are encouraging but hardly conclusive as yet. The blood pressures did not decline to normal levels but usually came down about 40 mm. hg. from maxima of approximately 220 mm. hg. They are convinced that their depressor substance is not the same as the one reported by Jablons, nor is it to be confused with any other depressor substance isolated in the past from the kidney, such as histamine, acetylcholine or adenosine. Its clinical use can only be expected to be of value in hypertension of renal origin.

Meanwhile Page (26) reported on the action of an extract made in his laboratory which supposedly contained the inhibitor earlier postulated by Helmer and Page (15). This extract brought on sharp reductions in blood pressure when injected in both hypertensive dogs and rats. In a report to the American College of Physicians in Boston in April, 1941, Page told of the effect of this extract on nineteen cases of malignant and essential hypertension seen clinically. The results were said to be favorable in all but two cases of malignant hypertension.

In conclusion one can only say that clinical application of the information learned from a study of experimental renal hypertension is still in the future. The work that has been done is promising and for the most part rests upon a firm experimental foundation. There can no longer be any doubt that experimental hypertension can be produced by renal ischemia. Furthermore, this hypertension is of humoral origin and arises as a result of the presence of an excessive amount of pressor substance in the circulation. This pressor substance acts directly on the arterioles to produce vasoconstriction. It is not, however, actually produced in the kidney but rather is a combination of a precursor found in the kidney and an activator already in the blood.

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CHORDOMA*†

A Case Report

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Chordoma may be described as a relatively rare, malignant, slowly growing neoplasm usually arising from remnants of the primitive notochord in the base of the skull or in the sacrococcygeal region of the spine.

EMBRYOLOGY

In man the notochord is the rod-shaped body of cells of ectodermal origin situated between the neural tube and the enteric tube. This structure forms early in the embryonic life and is the foundation of the axial skeleton. In the lower forms of life, for example, the Amphioxus, the notochord serves as the axial skeleton, but in man at the end of the fourth embryonic week it has almost completely disintegrated, with the exception of segments which remain and develop into the nucleus pulposus of the intervertebral disk of later life. During the process of disintegration and segmentation the adjacent masses of mesodermal cells grow in and surround the remnants of the notochord and the neural tube, from which the bony axial skeleton develops. The notochord extends the entire length of the axial skeleton of the embryo and traverses the area that later develops into the portion of the skeleton which extends from the base of the skull to the tip of the coccyx. This fact explains the presence of chordomas anywhere along the spinal column between these two points.

PATHOLOGY

The pathology of the chordoma was first fully established in 1858 by Müller and confirmed in 1894 by Ribbert. The origin of these tumors has been definitely proved to be the notochord. During postmortem examinations small jellylike masses of tissue have been found in the base of the skull just posterior to the pituitary fossa, which were first known as ecchondrosis physaliphora because they were thought to be areas of degenerating cartilage. Müller later identified these masses of tissue as vestiges of the notochord and now they are known as notochordal heterotopia. These masses of tissue are benign and must be differentiated from chordomas. Chordal cells may be found in or about any portion of the spine and may remain there as long as fifty years.

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Chordomas are firm elastic growths which enlarge and slowly exert pressure and invade nearby structures. The tumor remains fairly well encapsulated at times, but during the later stages breaks through and invades and destroys. One of its striking features is its affinity for invading bone. On one hand the base of the skull may be involved extensively while on the other the sacrum, the coccyx, and the ilia. Practically all of these tumors develop at the spheno-occipital synchondrosis of the base of the skull or in the sacrococcygeal region, and are about evenly distributed between these two locations. They may also be hypophysial, nasopharyngeal, maxillary, or vertebral. The tumor is generally considered to be rare, only 150 cases being reported up until 1935. Wood reported the first sacrococcygeal case in America in 1913. Daland reported the first American spheno-occipital case in 1919. These tumors may occur at any age, but the majority of the sacrococcygeal tumors occur in the fifth and sixth decades, while the spheno-occipital type occurs during the third and fourth decades. Why the spheno-occipital type occurs earlier in life has not been satisfactorily explained, although the intracranial lesion necessarily produces symptoms earlier. These tumors occur about twice as frequently in the male as in the female, the liability of the male to trauma being offered as an explanation for this fact.

The semitransparent, whitish or bluish gelatinous tissue of the chordoma is divided into lobules by fibrous trabeculae. The firmness of the tumor depends upon the amount of stroma present. The tumor is composed of cellular areas, the character of which resembles closely spindle cell sarcoma. Other areas are made up of large vacuolated cells filled with mucinous material. Intercellular vacuolation may be present as well as cytoplasmic vacuolation. The mucinous material being extracellular, the cell walls are indistinct and present a syncytiumlike structure. Differentiation of chordomas from colloid carcinoma, mixed tumors of the parotid, and myxochondroma is difficult on account of these features. Metastases occur in bones, liver, lungs, and the brain in from 40 to 60 per cent of the cases.

SYMPTOMS AND SIGNS

Because of the slow growth of these tumors symptoms occur over a prolonged period. The spheno-occipital type produces symptoms and signs of increased intracranial pressure, cranial nerve paralysis, and destruction of bone about the pituitary fossa. Roentgenologically, there are no characteristic signs. The diagnosis is invariably made at the postmortem table or by operative biopsy. The sacrococcygeal type produces pelvic pain, often sciatic in nature. The functions of micturition and defecation are disturbed. Obstinate constipation is a frequent symptom. Hemorrhoid-

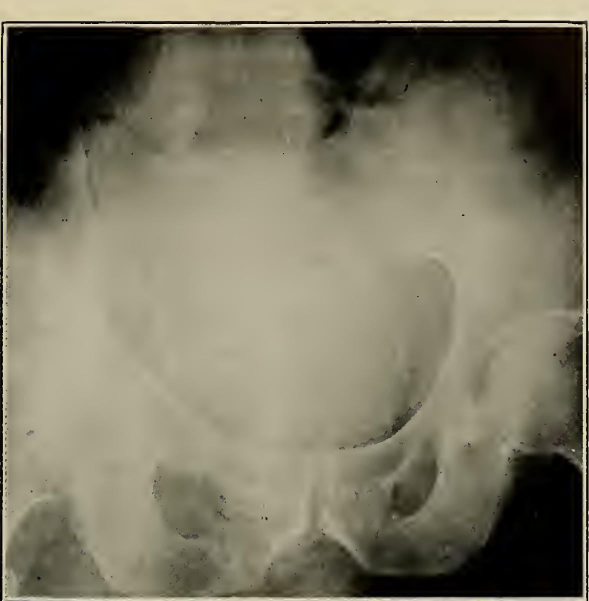

Fig. 1

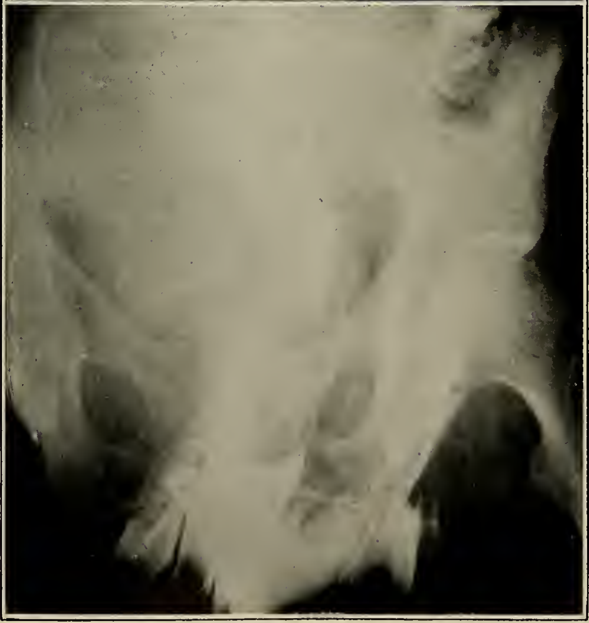

Fig. 2

Fig. 1. The roentgenogram shows a soft tissue mass in the pelvis with destruction of the lower three segments of the sacrum.

Fig. 2. A barium enema shows the rectum displaced to the left by the soft tissue mass. The rectum is also elongated and the anus is displaced markedly downward.

ectomy often precedes the final diagnosis. A tumor mass fills the pelvis, distorts and invades the pelvic viscera, and may protrude posteriorly in the region of the sacrum or sacro-iliac joints over which the skin moves freely. Hematuria and rectal bleeding may result owing to invasion, thus making the diagnosis difficult. The sacrum may be destroyed, the bone presenting a decalcified, irregular, reticulated appearance, declared by some to have typical characteristics. The diagnosis of the chordoma depends upon the location of the tumor, its slow growth, roentgenological appearance of the destroyed bone, and the microscopic studies.

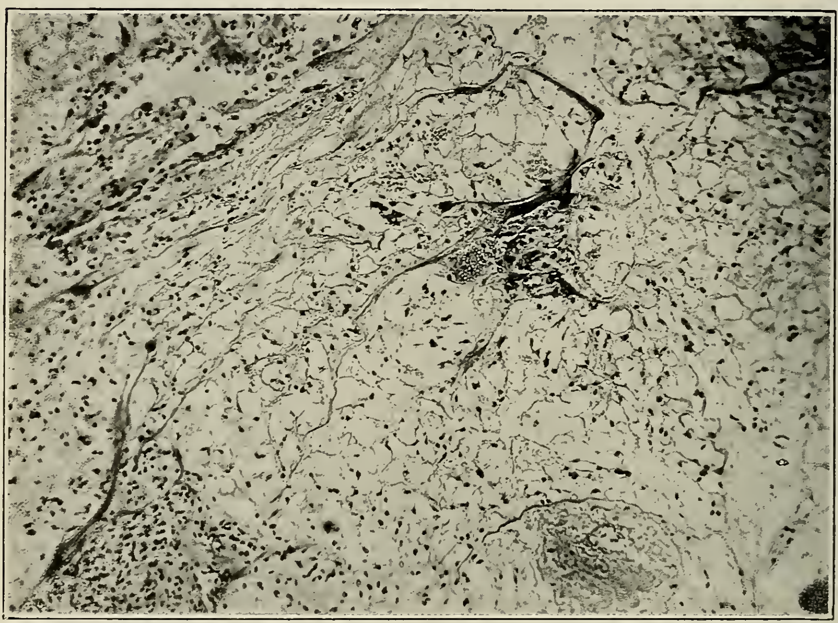

Fig. 3. Photomicrograph shows the large vacuolated cells filled with mucinous material. In areas definite cell boundaries are absent. Areas of hermorrhage are present and dense stroma is absent.

TREATMENT

Whenever possible the tumor should be removed surgically. This is frequently impossible on account of the invasion of vital adjacent structures. Chordomas are radioresistant because of the differentiated types of cells. Radiation is used alone or with surgery for palliation. Pain is usually relieved but the size of the tumor is rarely influenced. The outcome is invariably fatal, although life has been prolonged for several years after diagnosis.

REPORT OF CASE

H. M., a white male, age 60, came to consult a urologist on October 19, 1937 because of urinary difficulty. He had developed a gradually increasing dysuria for several months. He had also been severely constipated for the past three years, this condition becoming much worse during the past two months. His bowels moved only after drastic catharsis, at times assisted by pressure about the anus. Pain in the region of the right sacro-iliac joint had been present for about three months. This pain radiated down the right leg along the course of the sciatic nerve. A swelling was present in the region of the sacrum just to the right of the midline for the past two years, and had gradually become larger.

The physical examination showed moderate hypertension. A mass measuring about 6 cm. in diameter and protruding 3 to 4 cm. from the body surface was present in the region of the right sacro-iliac joint. The overlying skin was intact and freely movable. There was no discoloration and no inflammatory changes were present. The mass was quite firm, fixed, and not tender. A rectal examination showed the anus protruding markedly, with eversion of the mucous membrane. The gluteal fold was quite shallow. Digital examination revealed a large mass that filled the pelvis. The bladder possessed a retention of several hundred cubic centimeters of urine. A roentgenogram of the pelvis showed a marked destructive process involving the lower three segments of the sacrum and the coccyx. The texture of the involved bone presented a reticulated appearance. A barium enema showed a large pressure deformity of the rectum caused by an extrinsic mass, this segment of the large bowel being displaced outward and to the left.

The following day the patient was admitted to the University Hospital with acute urinary retention. After catheter drainage for a few days a biopsy of the sacral mass was done. Sections showed a growth composed of strands and alveolated masses of round and polyhedral cells. The nuclei stained deeply and the cytoplasm was vacuolated. In some places the stroma was delicate and scant, but in others it was more abundant. The findings were characteristic of chordoma but could be easily confused with colloid carcinoma or myxochondroma. Deep roentgen ray therapy was given through four ports of entry and amounted to a total dose of 8000 r units between October 28, 1937 and December 1, 1937. During this time a transurethral prostatic resection was done to provide more adequate bladder drainage.

The patient was discharged without complaint of pain. Bowel movements were less difficult though no definite change in the size of the mass could be made out by rectal examination.

The patient reported for examination on January 31, 1939 and at that time felt well. No urinary or rectal symptoms were present. The pelvic and sciatic pain had disappeared completely. The mass in the pelvis was much smaller.

Two years later he returned with a complaint of recurrence of pain in the sacral region which radiated along the course of the right sciatic nerve. He had gained thirty pounds. The bowels were slightly constipated. No urinary obstruction was present. The sacrum was slightly prominent and tender on the right. The anus was still protruding slightly.

Because of the recurrent pain the patient was given deep roentgen ray therapy again. A total of 7392 r units was divided among four pelvic portals between October 30 and November 27, 1939.

A roentgen ray examination of the pelvis on November 1, 1939 showed a slight regeneration of bone in the sacrum, but a large pelvic mass was still present.

By personal communication on February 24, 1941 the patient reported he was feeling well, except for an occasional pain in the sacrum. He is quite active and works every day.

SUMMARY

1. A case of sacrococcygeal chordoma is presented.
2. The history, signs, and symptoms are typical.
3. Deep roentgen ray therapy produced a partial regression of the tumor and alleviation of the symptoms.
4. Chordomas are radioresistant and should be removed surgically if possible.

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MULTIPLE MYELOMA WITH LOCALIZED AMYLOID DEPOSITS*†

A Case Report with Autopsy

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In 1937 Atkinson (1), reviewing the entire subject of multiple myeloma, recorded from the literature forty cases associated with amyloid disease. The first of these was reported by Adams and Dowse (2) in 1872 as a case of mollities ossium. The patient complained of increasing pain in the long bones of both upper and lower extremities. At autopsy lardaceous changes were noted in the liver and kidneys. The bone marrow contained a pink homogeneous gelatinoid substance which microscopically was found to consist of small spherical and oval cells having large nuclei.

Only a small number of the patients so afflicted had as presenting symptoms tumorlike amyloid masses. In four cases the tongue contained amyloid deposits; in two the amyloid apparently caused bowel obstruction; in twelve it was found in the myeloma tumors; in ten there were tumorlike masses about the shoulder and hip joints; in the remaining it was found in the arterial walls and the viscera.

Since Atkinson's review, Stewart (3) has reported a case in which nodules of amyloid were found attached to the ribs, scapulae, skull, and in the sheaths of the abdominal muscles. The diagnosis of multiple myeloma was based upon the finding of many plasma cells on bone marrow biopsy. An autopsy was not performed.

More recently Tarr and Ferris (4) have reported a case with autopsy findings. Amyloid masses were noted in front of each ear, in the anticubital fossae, and on the lateral borders of the scapulae. At autopsy the marrow of the sternum and the vertebrae was found to be almost entirely replaced by myeloma cells.

Most of the reported cases had certain prominent clinical features, namely, progressive weakness, loss of weight, stiffness of the joints, and inconstant deep-seated pain. Physical examination revealed amyloid nodules, the limitation of motion in various joints, emaciation, and hepatomegalia. The commonest significant laboratory findings were hypochromic anemia, Bence-Jones proteinuria, hyperproteinemia, and positive Congo red tests for amyloid. Roentgen ray examinations revealed discrete areas of bone destruction in the skull, ribs, clavicles, humeri, femurs, and iliac bones.

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At autopsy myeloma cells were found in the bone marrow. There were masses having the staining characteristics of amyloid throughout the body. Focal scarring was evident in the renal cortex. Pink albuminous precipitates were noted in the convoluted tubules and homogeneous casts, and in the straight collecting tubules.

The rarity of the syndrome and the puzzling diagnostic problem that it presented warrant reporting the following case.

CASE REPORT

The patient, a 53 year old white male farmer, was admitted on the medical service of the University Hospital on February 19, 1938. He had been well until June, 1937, when he noticed painful swollen areas in both groins. This persisted until November, 1937, at which time he developed pneumonia in the lower lobe of the left lung. During convalescence his right leg became greatly swollen for a period of three to four weeks. A dry cough and anorexia persisted. The swollen areas in the groins steadily grew larger. The patient felt progressively weaker and lost weight to the point where hospitalization was advised.

The family history, past history, and review of the systems were important only in that the patient had a mild pneumonia five years prior to this time.

On admission, physical examination revealed a generalized pallor and looseness of the skin. The spine was flexed slightly to the right, the shoulders were carried forward, the thighs were slightly flexed, and the knees were spread apart. The pupils were round, central, equal, and reacted to light and accommodation. Deviation to the right was noted in the nasal septum. The teeth were worn and carious. The tonsils were atrophic. The cervical and epitrochlear lymph nodes were not enlarged. There were a few small nodes palpable in the right axilla. Distinct pulsations were evident in the carotid and brachial arteries. The isthmus of the thyroid was somewhat enlarged. A freely movable mass about the size of a walnut and having the consistency of cartilage was apparently attached to the inferior angle of the right scapula. Expansion of the chest was limited but the lungs were otherwise clear. The heart was not enlarged and the sounds were of normal intensity. The blood pressure was 120 mm. of mercury systolic and 62 mm. diastolic. The liver and spleen were not felt. The external genitalia were normal. Rectal examination revealed a fibrous prostate. In each inguinal region were bony, hard spherical masses measuring about 8 cm. in diameter. These were apparently fixed to the pubic rami and displaced the femoral arteries anteriorly and medially. Extension and external rotation were limited at both hips. There was moderate limitation of motion in all directions of the cervical and thoracic spine and of abduction and external rotation of the left humerus.

Laboratory findings. The urine was persistently acid, had a specific gravity ranging from 1.008 to 1.018, contained traces of albumin, occasional coarsely and finely granular casts, and the Bence-Jones protein.

The two hour excretion of phenolsulphonphthalein was 35%.

Examination of the blood showed 2,770,000 red blood cells; 3,450 white blood cells; segmented polymorphonuclear neutrophils 80%; nonsegmented forms 3%; lymphocytes 15%; monocytes 2%. The color index was 0.8. There was moderate anisocytosis and poikilocytosis.

Examinations of the stools revealed nothing of importance.

The serologic test for syphilis was negative. On heating to 56 C. the serum appeared chalky.

The blood chemical examination revealed the following: nonprotein nitrogen 66 mg. %; creatinine 3.2 mg. %; sugar 109 mg. %; calcium 10.9 mg. %; phosphorus 5.1 mg. %; cholesterol 200 mg. %; chlorides 335 mg. %; total fatty acids 469 mg. %. The albumin-globulin ratio was 4.45/4.63. The van den Bergh reaction was of the prompt direct variety with only a trace of bilirubin.

The blood sedimentation rate was 18 mm. in one hour.

The basal metabolic rate was +5%.

The fractional gastric analysis revealed the presence of free and combined hydrochloric acid in normal amounts.

Roentgen ray examinations of the chest, right scapula, left shoulder joint, right and left femurs, skull, kidneys, ureters, and bladder showed no abnormalities. The lumbar spine showed slight hypertrophic changes. Small punched-out areas of bone destruction were noted at the head of the left first rib, on the iliac crest close to the spine on the left, and on the outer aspect of the left acetabulum.

On February 26, 1938 the mass in the right subscapular region was excised for biopsy. On March 7, 1938 a portion of the mass in the right inguinal region was removed for the same purpose. Grossly the masses consisted of moderately firm tissue which on sectioning exhibited a glistening gray cartilaginouslike surface and which turned black on treatment with Lugol's solution and sulphuric acid. Microscopically the sections showed an amorphous eosinophilic tissue displaying little fixed structure, through which were scattered spindle-shaped fibroblastic nuclei and thin-walled capillaries.

On March 9, 1938 a Congo red test revealed 23% absorption of the dye. This test was repeated on April 12, 1938 and revealed no absorption of the dye. The presence of amyloid disease was, therefore, not confirmed by this particular test.

The punched-out areas shown in the roentgenogram were inaccessible for biopsy by aspiration. A sternal puncture was therefore performed, but the marrow obtained showed only an occasional plasma cell. Repeated examinations of the blood for plasma cells were of no avail.

A high caloric diet with added vitamins and iron was prescribed for the patient. He was given roentgen ray therapy to the pelvis, right first rib and the parathyroids from March 15, 1938 to March 26, 1938. Blood transfusions were given at intervals of two to three weeks. Potassium iodide up to 90 gr. a day was administered. The general condition of the patient did not change. His temperature was higher than normal, at times reaching 100 C. The masses did not change in size or shape. The posture and strength remained the same. Repeated roentgenograms of the involved areas revealed no progression of the lesions. By May 4, 1938 the blood nonprotein nitrogen which was 66 mg. % on admission had dropped to 25 mg. % and the albumin-globulin ratio which was 4.45/4.63 had changed to 4.49/2.85. The red blood cells at this time numbered 2,500,000, the white blood cells 1,700, with the differential showing segmented forms 47%, nonsegmented forms 6%, lymphocytes 41%, eosinophils 2%, and monocytes 4%. The color index was 0.78. The patient was discharged on May 15, 1938.

During the ensuing seven months at home the patient became progressively weaker and more dyspneic. He developed nodules at both sternoclavicular joints and along the styloid processes of both radiuses. The nodules slowly enlarged and became painful. Toward the end of December, 1938 he developed a febrile illness with cough, and died on December 30, 1938.

Necropsy: The autopsy was performed with limited facilities at an undertaking establishment.

The body was that of an emaciated middle-aged white male and measured 160 cm. in length. The pupils were round, equal and regular. There were two spherical masses at the sternoclavicular joints, each measuring about 2 cm. in diameter. Similar nodules were noted about the styloid processes of the radiuses. In each groin there was a large round stony-hard mass measuring 8 cm. in diameter. Overlying the mass in the right groin was a longitudinal scar resulting from a previous biopsy. The teeth were markedly carious.

In the thoracic cavity there were dense fibrous adhesions about the apex of each lung.

The heart weighed 350 gm. On sectioning, the attached portion of the aorta showed moderate atheromatous changes. There was a yellow and pink mottling of the endocardium on the upper half of both ventricles.

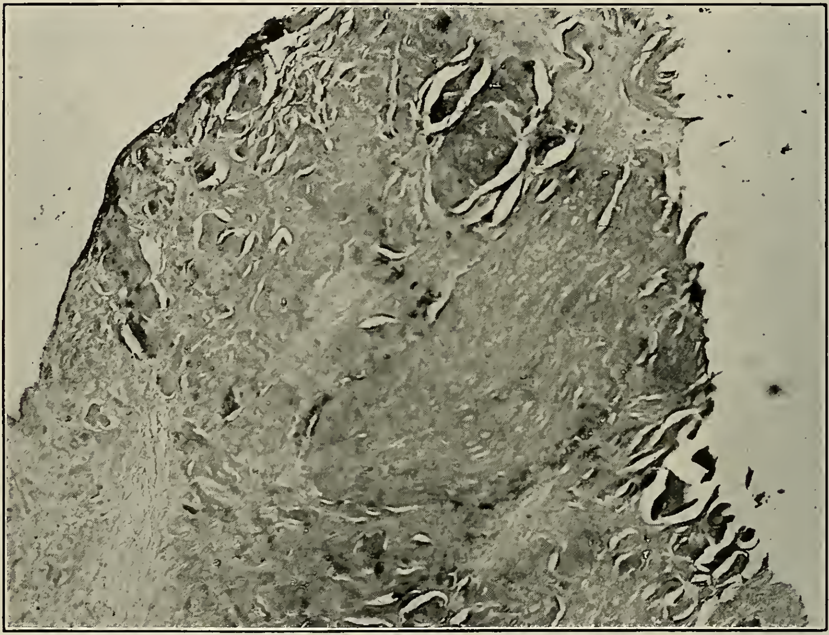

Fig. 1. Amorphous eosinophilic tissue displaying little fixed structure and scattered fibrous strands typical of amyloid.

The left lung weighed 650 gm. and the right 760 gm. There were a few fibrous tags attached to the apex of the left lung. The dependent portions of both lungs were somewhat darker and firmer than normal. Large quantities of clear, frothy fluid could easily be expressed from these regions.

The liver weighed 1,500 gm. On sectioning, the cut surface revealed a typical nutmeg appearance.

The spleen weighed 150 gm. and was wrinkled and doughy. On sectioning, the cut surface had a dark red color. The structure was poorly preserved.

The pancreas and adrenals presented no abnormalities.

The kidneys were somewhat softer than normal. The surface presented irregular white mottled areas in which were scattered reddish flecks. There were several small cortical cysts. On sectioning, the cut surface appeared pale. The structure was not well defined.

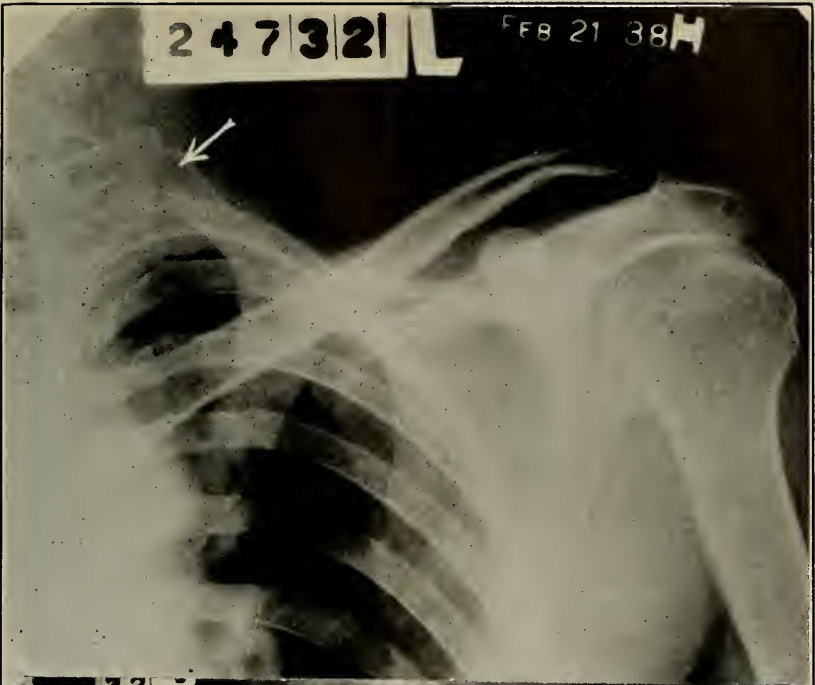

Fig. 2

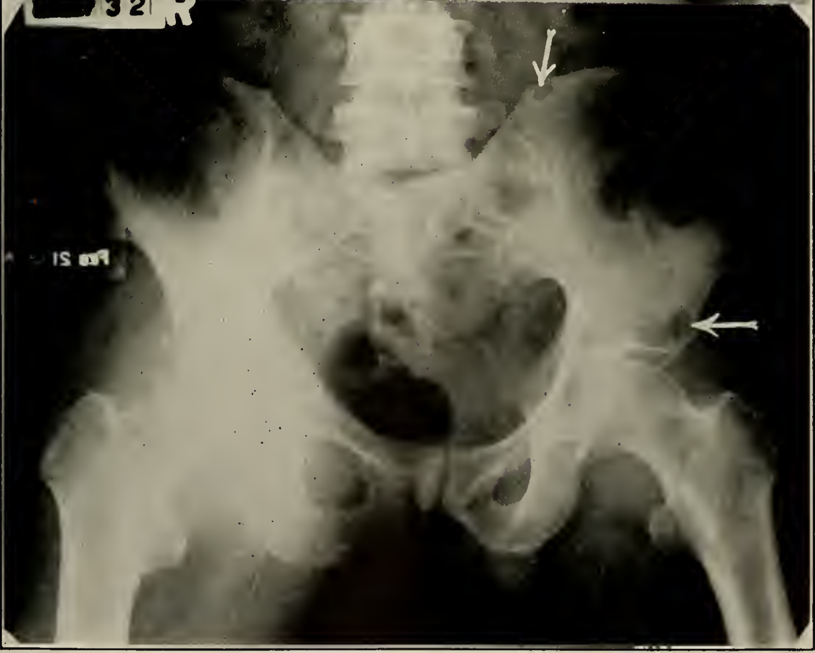

Fig. 3

Fig. 2. Punched-out areas of bone destruction, head of the first left rib.

Fig. 3. Areas of bone destruction, the left iliac crest and outer aspect of left acetabulum.

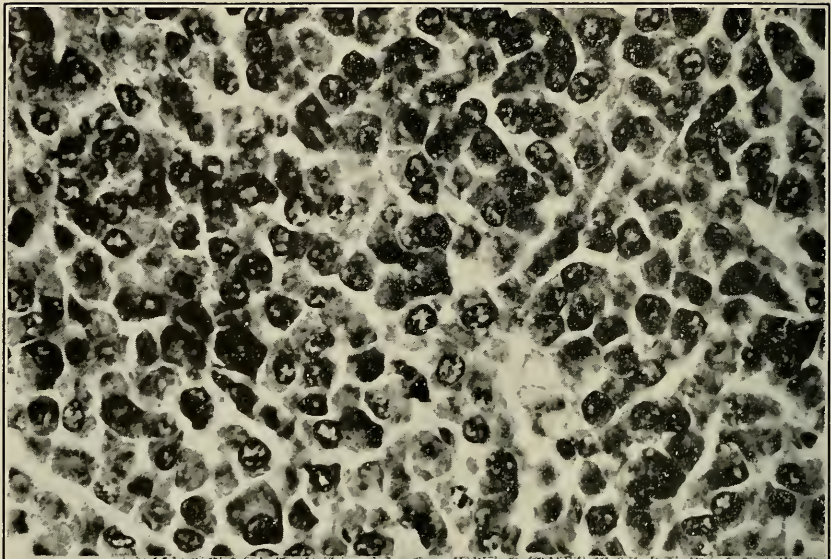

Fig. 4

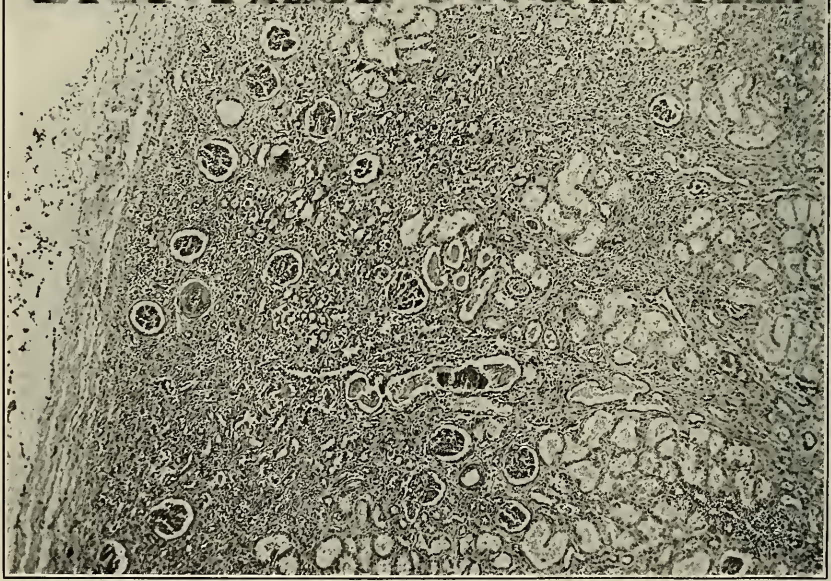

Fig. 5

Fig. 4. Myeloma cells replacing sternal marrow.

Fig. 5. Scarred and hyalinized glomeruli, albuminous precipitate in lumina of convoluted tubules. Hyaline cast in the center.

The inguinal masses were well circumscribed, white and waxy, and were apparently attached to the bone.

A section through the manubrium showed a soft grayish-pink marrow in which was a small soft yellow nodule.

Microscopic examination. Heart: Deposits of acidophilic material (amyloid) were evident in the small coronary arteries and there was a definite increase in the transverse fragmentation and scarring of the myocardium.

Lungs: Amyloid deposits were noted in the small arterial walls. There was marked pulmonary edema, with scattered areas of bronchopneumonia.

Liver: The central zones of the lobules were atrophic. The sinuses in these areas were considerably distended. Many of the liver cells appeared degenerated and had pyknotic nuclei. No amyloid deposits were observed.

Spleen: The capsule was somewhat distended. The trabeculae were more numerous than usual and seemed somewhat hyalinized. The pulp was unusually cellular and contained large quantities of pigment. The malpighian bodies showed no evidence of germinal activity. There were no amyloid deposits.

Kidney: The section showed many obliterated and hyalinized glomeruli with corresponding atrophic tubules (Fig. 5). Many of the tubules contained a deeply staining acidophilic material which might be considered to be the Bence-Jones protein. There was no evidence of amyloid deposition.

Inguinal mass: The section showed an amorphous eosinophilic material, with scattered spindle-shaped nuclei typical of amyloid.

Bone Marrow: The section showed a nodular area in which all of the cells appeared to be plasmablasts arranged in a sheetlike fashion, the typical picture of multiple myeloma (Fig. 4).

Anatomic diagnosis. Multiple myeloma; amyloid deposits, inguinal regions, styloid processes of radiuses, sternoclavicular joints; pulmonary edema; bronchopneumonia, bilateral; chronic passive congestion, liver; fat degeneration, endocardium.

DISCUSSION

On admission to the hospital the patient presented a puzzling diagnostic problem. The character of the stony-hard masses in each inguinal region could not be determined until sections obtained at biopsy displayed the characteristic staining reactions of amyloid (Fig. 1). The appearance of a precipitate on heating the blood serum to 56 C. and the presence of the Bence-Jones protein in the urine suggested some bone-destroying lesion as the common etiological factor for both amyloid deposition and Bence-Jones proteinuria. The demonstration of punched-out areas of bone destruction in the head of the left first rib and in the pelvis (Fig. 2 and 3) suggested a chronic suppurative process, metastatic neoplastic disease or multiple myeloma.

The involved areas of bone were inaccessible for biopsy. A primary site for a neoplasm could not be demonstrated. The similarity of the course of this case with those of reported cases of multiple myeloma associated with localized amyloid deposits was recognized. A similar diagnosis was therefore made at the time of the patient's discharge. This was not confirmed, however, until typical myeloma cells were demonstrated in the bone marrow at the time of autopsy.

SUMMARY

A case of multiple myeloma with localized tumorlike amyloid masses as presenting symptoms is herewith described. It is the thirteenth case of this type of amyloidosis reported to date.

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BRONCHIAL ASTHMA IN THE PRINTING TRADE—NONOFFSET SOLUTIONS*†

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For many years acacia has been widely used in medicine and industry and has been considered completely innocuous. In fact, relatively large quantities have been used intravenously in thousands of cases without serious reactions (1). However, a few years ago rapidly drying materials containing acacia were introduced into the printing trade as nonoffset solutions. These are sprayed onto the printed sheet, where they dry rapidly and leave a thin transparent film which serves two important purposes. First, it prevents smearing when the next sheet is placed upon the preceding one; secondly, it prevents the printed letters from appearing on the reverse side of the uppermost sheet. This rapidly drying spray creates a finely divided dust that would seem, without question, to be entirely harmless.

Shortly after the adoption of this procedure pressmen complained of a sensation of tightness in the throat and an investigation was made by Smith and Burke (2) upon the request of a pressmen's union. They concluded that the materials used in the spray were essentially innocuous and that the symptoms mentioned were transient and harmless. However, soon after their attention was called to a case of bronchial asthma which was proved to be the result of the practice previously mentioned.

Upon searching the literature they found only one other case which occurred in a plaster molder (3). Furthermore, they tested thirty allergic individuals with entirely negative results, thus proving that acacia sensitivity is not common in allergic patients.

Humperdinck (4) studied sixty-six employees of a printing plant employing this method. He estimated that in eight hours from 380 to 500 grams of acacia were used in the operation. The employees examined included thirty-four male printers, two male helpers, sixteen female feeders, and fourteen women who removed the treated sheets. He found two types of symptoms: a larger group who complained of bronchitis newly acquired or aggravated by the spray, of catarrh, emphysema, and an increase of fibrous tissue revealed on roentgen ray examination; the smaller group consisted of two cases of asthma, one of which was of true occupational origin. Two cases of eczema were encountered and he reported that the blood eosinophils were found to be increased in many cases. Apparently,

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no skin tests were done as none are mentioned. Needless to say, it is difficult to evaluate this rather incomplete report. It is significant, however, that with sixty-six exposed persons he found only one case of true occupational bronchial asthma.

The experience of the authors is in accord with that of these observers. A study was made of twelve workers in the firm which employed the patient to be discussed in this paper. It was found, as will be described later, that no other cases had occurred in the same plant and that none of the other employees exposed to the spray exhibited positive skin tests to acacia upon intradermal testing. The extract used was one that gave strongly positive tests upon directly testing the patient and upon passive transfer tests using his serum.

The foregoing seems to suggest that acacia is not an active sensitizer. However, an article (5) published after the completion of this report cites ten cases of asthma resulting from the inhalation of acacia. Unfortunately, the total number of persons exposed in the combined plants concerned is not stated and it is impossible to gauge the percentage of sensitizations resulting. From the material published it would seem that asthma from acacia must be considered a true occupational disease even though the frequency of its occurrence cannot be determined from the data available.

Because of its possible economic importance under the different state compensation laws, some of which include occupational diseases among the compensable conditions, it was felt this case warranted reporting. This opinion was confirmed by casually questioning numerous practitioners, none of whom had any knowledge of this condition as a possible clinical problem. It would seem that asthma caused by this substance would most certainly be compensable in view of the fact that its etiological significance is so readily susceptible to incontrovertible proof.

CASE REPORT

BRONCHIAL ASTHMA—ACACIA

The patient, a white male, 31 years of age, was seen on March 25, 1940 with a complaint of bronchial asthma. His family and past history were without significance both from an allergic and a general standpoint.

Present illness. The symptoms began about two years ago with shortness of breath which came on gradually and was accompanied by cough. There was no initial respiratory infection at the time of onset. The symptoms rapidly reached their maximum and continued at that level. The attacks occurred approximately every seven days and were of about a week's duration; they continued day and night, with more severe symptoms nocturnally. Between attacks his freedom from dyspnea was relative rather than absolute, as he had a definite amount of dyspnea nocturnally even then, although he did fairly well in the daytime while at work. Usually he was able to force himself to work in spite of these symptoms. He was seen by Dr. L. Serra and referred for study.

Occupation. For the past eleven years he has worked for his present employer as a pressman. About five years ago a new spraying apparatus intended to prevent offset effects in printing was installed to replace the former method which relied upon heat to accomplish this purpose. For months he experienced no difficulty. Then the dyspnea began, although the patient did not connect it with his work in any way. In fact, he reported that he was worse while at work because he was more active and explained that increased activity at home would have the same effect. At no time did he suspect the fluid used in his work.

Seasonal variation. There was no variation with the seasons, according to the patient.

Geographic. One day spent in Upper Darby, Pa. was without symptoms, but he had been asymptomatic when he left home. No other trips were taken.

Local changes. The patient felt worse at bedtime; no other information of possible significance was revealed.

Food. The symptoms were not affected by food at any time.

Animals. He had a dog for fifteen years, canaries for three years. Feather pillows were used because an elimination for trial had been without result. No adverse effect from animal contacts was noted by the patient.

Physical examination. This disclosed nothing of interest with the exception of slight hyperresonance over the lungs, prolongation of expiration, and musical râles over the bronchial trees throughout both phases of respiration.

General laboratory procedures. A blood eosinophilia of 12% was revealed with all other findings negative, including urinalysis, nasal smears, and sputum examinations.

Intradermal skin tests, using 0.02 cc. of the extract, gave significant results. Of the inhalants used, grass and ragweed pollen extracts (4% in 50% glycerine) in a 1:10 dilution were strongly positive in the cases of Bermuda grass, June grass, red top, and orchard grass. The animal epidermals were all negative. Orris root was strongly positive objectively, 3 plus, but as in the case of the pollens there was no subjective itching. Numerous foods gave positive reactions also, but in none of these instances could a symptom causation relationship be shown.

The patient brought in a supply of the nonoffset solution (concentrated). An intradermal test was quite positive, causing subjective itching as well as objective whealing and erythema. Passive transfers were strongly positive at the test site containing the patient's serum but completely negative at the control site tested. As his employer had decided to change to a new product containing dextrin rather than acacia, this was tested in exactly the same way with completely negative results.

Clinical course. During the duration of this study the patient fared badly and this resulted in several forced absences from his work. In each instance he became clear of symptoms after two or three days and remained so as long as he stayed away from work. Upon returning to work there was a recurrence of symptoms after from twenty-four to forty-eight hours.

Treatment with a sterile preparation of the nonoffset solution was started and given in a routine manner. Because of the severity of his symptoms he was forced to stop work until a concentration of the material could be reached in treatment that it was hoped would be therapeutically effective.

In June, 1940 gauze surgical masks were tried; after a few weeks they were discarded as ineffectual. In October, after several months of idleness, the patient was advised to return to work as it was felt that he had received sufficient treatment to warrant a trial. At the same time his employer obtained a standard mask for him from one of the usual commercial sources. According to the manufacturers, this was intended for use "in low concentrations of acid gases and organic vapors such as acetic acid and others."

Either as a result of treatment, the mask or, more probably, both, he remained symptom-free upon returning to work and has had no recurrence. Late in 1940 the acacia

TABLE 1

CASE	ALLERGIC FAMILY HISTORY	ALLERGIC PAST HISTORY	INTRACUTANEOUS TESTS						
			1:1000 Acacia*		1:20 Acacia†		N. O. Sol. (Conc.)‡		Symptoms
			Subj.	Obj.	Subj.	Obj.	Subj.	Obj.	
1	Neg.	Neg.	Neg.	Neg.	Neg.	2+	Neg.	2+	No dryness of nose and throat. No asthma or dyspnea.
2	Neg.	Neg.	Neg.	Neg.	Neg.	2	Neg.	2+	Dryness. No asthma or dyspnea.
3	Neg.	Neg.	Neg.	Neg.	Neg.	1+	Neg.	2	No dryness. No asthma or dyspnea.
4	Neg.	H.F. 7 yrs., free 3 yrs.	Neg.	Neg.	Neg.	1+	Neg.	2	Dryness. No asthma or dyspnea.
5	Neg.	Neg.	Neg.	1	Neg.	1+	Neg.	2	No dryness of nose and mouth. No asthma or dyspnea.
6	Neg.	Neg.	Neg.	1	Neg.	1+	Neg.	2	No dryness of nose and mouth. No asthma or dyspnea.
7	Neg.	Neg.	Neg.	2	Neg.	1+	Neg.	2	No dryness of nose and mouth. No asthma or dyspnea.
8	Neg.	Neg.	Neg.	Neg.	Neg.	Neg.	Neg.	1	No dryness of mouth and nose. No asthma or dyspnea.
9	Neg.	Neg.	Neg.	1+	Neg.	Neg.	Neg.	1+	No dryness of nose and mouth. No asthma or dyspnea.

N. and M. (nose and mouth) represent minor effects.

A. and D. (asthma and dyspnea) represent major effects.

* The concentrated acacia solution was 5%, from which the test dilutions were made.

† The 1:20 dilution of acacia caused burning at the test site and an irritative reaction. This was obviously not a truly positive test in any test subject. The negative tests with the 1:1000 solution verify this.

‡ The nonoffset solution in no instance caused itching in the control cases. It did cause subjective symptoms at the site of inoculation in the patient and also upon passive transfer with the patient's serum. The control site was negative in the passive transfer recipient's skin.

TABLE 1—*Concluded*

CASE	ALLERGIC FAMILY HISTORY	ALLERGIC PAST HISTORY	INTRACUTANEOUS TESTS						
			1:1000 Acacia*		1:20 Acacia†		N. O. Sol. (Conc.)‡		Symptoms
			Subj.	Obj.	Subj.	Obj.	Subj.	Obj.	
10	Neg.	Neg.	Neg.	2	Neg.	1+	Neg.	2	Dryness of nose and mouth. No asthma or dyspnea.
11	Neg.	Neg.	Neg.	Neg.	Neg.	1+	Neg.	2+	Dryness of nose and mouth before acacia spray was used. No asthma or dyspnea.
12	Neg.	Neg.	Neg.	Neg.	Neg.	1+	Neg.	2	Negative
	None	1 case 8% pos.	None	40% pos.	None	80% pos.	None	100%	N. and M. 32% pos., A. and D. none pos.

solution was discarded and replaced by one containing dextrin. All treatment was then discontinued and he has since discarded the mask without adverse effect.

In an effort to determine the frequency of induced sensitivity in pressmen exposed to acacia in this fashion, about 50 per cent of his fellow workmen, who were also exposed to the solution, were studied. The findings are shown in table 1.

In questioning the control subjects, two types of symptoms previously described were sought: first, evidences of local irritative reactions represented by cough, nasal and throat symptoms, and a vague sense of oppression in the substernal region as described by other observers; secondly, frank bronchial asthma. In four cases, dryness of the nose and mouth were admitted which began, apparently, at the time that the acacia solution was introduced into the plant. There was no complaint of bronchial asthma. Case No. 4 had had hay fever up to three years previously and was considered to be atopic: however, frank allergy to acacia could not be demonstrated as he had never had asthma or other symptoms that might be ascribed to his work.

It will be noted in table 1 that of the four individuals tested who complained of minor effects only one, No. 10, responded with any degree of positivity to intradermal testing with 1:1000 acacia extract. Even this test was unassociated with any subjective symptoms, such as itching, at the test site. In comparing the group as a whole it was revealed that objective reactions with this dilution occurred in 40 per cent of the cases, whereas in the group showing minor effects only 25 per cent responded.

Apparently, the minor effects could not be considered as the result of specific sensitization induced by exposure. The authors agree with Smith and Burke (2) that the minor effects represent local irritative manifestations and not systemic, allergic sensitivity.

SUMMARY

A brief review of the literature of bronchial asthma caused by acacia found in nonoffset solutions used in printing is presented. That it occurs frequently, as is stated by certain writers, seems to be untrue. A clear-cut case is reported which obtained complete relief from therapeutic measures. Later, elimination of the substance from his occupational environment resulted in an apparent cure. A group of his fellow workmen was studied to determine the frequency with which adequate exposure resulted in true sensitization with the production of frank bronchial asthma. Minor effects were correlated with acacia skin tests and no relationship was revealed.

CONCLUSIONS

1. Acacia is a potential sensitizer when there is adequate exposure to the substance as an inhalant.
2. Bronchial asthma may be induced in those persons exposed who exhibit a predisposition to such sensitization.
3. Despite the statements in the literature to the contrary, bronchial asthma is infrequently produced by this exposure.
4. When bronchial asthma does result from adequate acacia contact it is an occupational disease and should be so considered in laws governing compensation for industrial accidents and illnesses.
5. The minor effects resulting from nonoffset sprays, namely, upper respiratory tract dryness with occasional cases showing evidences of inflammation, represent irritative phenomena rather than specific sensitivity to acacia.

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THE PATIENT*†

LOUIS H. DOUGLASS

BALTIMORE, MD.

For one person to offer advice to another person or group of persons has always seemed to me to be quite presumptuous, and a practice not to be indulged in except under most unusual circumstances. In the only field in which I might be at all qualified to give advice, the field of medicine, very little advice is needed. The career for which you have been preparing yourselves and upon which you are about to embark is, without question, the most fascinating one that exists today. It makes little or no difference which branch of medicine you choose to follow, whether it be general practice or one of the specialties, you will find it intensely interesting and absorbing. Medicine is a harsh task-mistress, demanding much from us, her pupils, in the way of work, sacrifice of pleasure, and study, but she is generous in rewarding her industrious pupils, with a reward that all too many men who lead a less Spartan existence miss, the reward of complete absorption and satisfaction. Each patient presents his individual problem or problems and each day brings something new to be learned and stored up, sometimes as an addition to what we already know and sometimes as a replacement for another piece of information which in turn has to be discarded. You will be constantly striving toward an unattainable goal, the goal of complete knowledge. It should be your daily and humble prayer to the gods that be that that goal remain forever unattainable, for should you, by any chance, feel that you have reached it, then your career is ended. The incentive and the urge are gone and you have no reason to carry on further.

In the practice of medicine we are dealing with an extremely complicated piece of mechanism, the human being, and it is of this mechanism which presents itself to us as *The Patient* that I wish to speak this evening. Perfect health has been described as that state of complete unawareness of the functioning of any part of the body, and a person enjoying perfect health has no reason or desire to consult a physician. But when some derangement of this complicated mechanism occurs and the individual becomes aware of one or more of the functions of the body, and especially when that awareness manifests itself as pain, then assistance is likely to be sought and citizen John Smith becomes patient John Smith. For to whom can an ailing person turn for sympathy, kindness and relief, other than to him whose aim and purpose in life it is to be always ready and always prepared to furnish this same sympathy, kindness and relief? And the patient who has come to us because of some abnormality, and although in many instances a complete stranger has intrusted to us his well-being, possibly his life itself, has a perfect right to be particular and to demand assurance that his choice is a wise one.

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* Address delivered at the Pre-Commencement Exercises, School of Medicine, University of Maryland. The Lyric, Baltimore, June 6, 1941.

† Professor of Obstetrics, School of Medicine, University of Maryland.

The patient, it should be remembered, is extremely interested in one thing and only one thing when he comes into your office, and that one thing is himself and his personal welfare. He cares nothing about the interests or troubles of his doctor; indeed, the doctor is supposed to be a very superior sort of person who is not subject to trouble, fatigue or any of the diseases or misfortunes which afflict ordinary persons.

Although exacting in his demands and rightly so, the patient is usually appreciative and grateful for two things he receives all too rarely, a deep and attentive interest in him and his problem, and honesty. Too often a patient goes to a doctor and is received with scant courtesy, not being allowed to tell his story completely, or if allowed he is not listened to with any degree of sympathetic attention. The doctor is busy, has other patients waiting for him, is overdue for an operation or a meeting, or for some other reason is unwilling to devote the time necessary to this particular individual. The result is that the patient leaves his office dissatisfied and discouraged. No matter how brilliantly correct a diagnosis has been made, nor how proper the treatment or advice given, little good can come from it for that one important ingredient to recovery, confidence, is lacking. Or, and this is still more important, the physician may not be entirely honest with the patient. By this I do not mean the performance of unnecessary operations or the unwarranted use of various physiotherapeutic or other devices to increase his income. I mean that the doctor may paint a picture which is not quite true in order that his reputation may be enhanced should the patient recover. He may do the opposite and minimize the gravity of the condition for fear that the patient might become alarmed and go to someone else. These are only two ways in which an otherwise reputable physician may be slightly dishonest. I am sure that others will occur to you.

Pursuing this same line of thought we might ask whether it should ever be admitted to a patient that we don't know the answer to a question he has asked us about himself and his condition. To me the answer to that is simple: why not? No one doctor can possibly know all there is to know about medicine, and even to the wisest there must at times come a question to which he does not know the answer. It is, of course, gratifying to be able to reel off a glib explanation, but if we don't know isn't it better, wiser and more honest to frankly say we don't, rather than to try to fool the patient with some involved combination of words and phrases which means little to him and less to us? Just say that we don't know but that we will try to find out and supply the information at a later date. When we are fortunate enough to know the answer, isn't it better to give it in language the patient can understand and appreciate, rather than in deeply technical words and phrases which we know are beyond his comprehension?

Again there are unpleasant facts which, as physicians, we are compelled to tell patients and their families, and it is my feeling that when the occasion arises they should be told gently, yet frankly and honestly; time should be taken to make all necessary explanations and to allow for whatever reactions may ensue, to subside. One should assume the position of the recipient, and should conduct himself with patience, sympathy and tact. Indeed, it would probably be excellent training for every physician if at an early period in his career he could be a patient himself, with a fairly major condition, and in this way obtain the viewpoint of the "other side." Obviously, it would be impossible for every physician who is specializing to be a patient in his own specialty but, on the other hand, this is not necessary, for the essentials of physician-patient relationship are constant in all branches of medicine and the desired end would be obtained.

Any remarks at this time when we are facing a national crisis should certainly include some reference to the obligations we owe to this, Our Country. The reference this evening is going to be brief, for I have a supreme confidence in the youth of today and especially that portion of it represented here. There is a perfectly understandable desire on your part to complete your training in medicine and to start upon your life work, and you also quite naturally resent anything which interferes with your plans. On the other hand, you must be fully aware that our entire scheme of life is being threatened, the very foundations upon which our country rests are being undermined, and there is a grave danger that we may none of us be allowed to do as we have done in the past and lead a free and independent life. That is, unless we do as our forefathers did and fight for those things we cherish so highly.

Again, a crisis of this kind can last only a comparatively short time; the fires are burning too fiercely and must of necessity soon subside. And so the time required of any of us to give to the protection of our own well being, our own interests and our own ideals is bound to be short in the course of a lifetime. And we are more fortunate than some others in that while we are serving our country we are still engaged in the practice of medicine, we are still able to add to our knowledge, and we are still handling and treating patients.

As I said before, I have complete confidence in each and everyone of you and I am sure that when the time comes you will do your duty and will not consider it a sacrifice but your inalienable right as an American to fight your own fight, and to insure the safety and freedom of yourself and your family.

A grave responsibility will descend upon your shoulders when you graduate, complete your hospital or postgraduate training, and finally begin the practice of your profession, the responsibility of the life and well being of those who entrust themselves to your care. You will become the repository of confidences, often of a most personal nature; you will have to act not only as medical but as spiritual adviser. You will hold in your hands the power to bring life into the world, to guard the health not only of your patients but of the community as a whole, and you will often be called upon to make the inevitable passing of a life a little less terrifying and distressing. The troubles of your patients will be brought to you and laid in your lap for you to solve, with an implicit trust that what you do will be wise and just. Can you do all of these unless you make a special exhaustive study of that complex, often suffering and frequently bewildered specimen, *The Patient?*

One of the great disadvantages of the modern practice of medicine is that we may come to look upon patients as cases and not as individuals. This is probably the result of the emphasis of recent years on scientific research and the application of proved experiments in the laboratory to the patient. Here the interest in the result is so great that there is a tendency to overlook the fact that we are dealing with a human being and to appear to be aloof and apparently indifferent to any reaction not connected with either the disease or the treatment. It is by no means my intention to decry or to minimize research, for it is the basis of most of the advances which we have made and are continuing to make, and more research should be done. On the other hand, patients should not be made into or treated as laboratory animals, with charts, venipunctures, weighed and calculated diets, and no personal contact or word of explanation from the doctor as to what is going on or what their chances are for recovery.

In the minds of each of you this evening there is, or at least there should be, one main idea—success, and no one could be more sincere than I in wishing you this same success, and in large amounts. May I, however, point out that there are various kinds of success; the first, that manifested by monetary return is fairly easy to obtain, if your goal be not too far away. Then we have success indicated by widespread fame, so that you become known nationally or perhaps even internationally. And finally there is the success which results from the satisfaction of a difficult job well done, the success of a peaceful mind and an undisturbed conscience. This is the kind of success which I wish most heartily each and every one of you will attain, and in the final analysis it is based on this very *Physician-Patient* relationship of which I have been speaking. For it is when patients come to you, and having come once return time after time with confidence and faith that you will care for them honestly and conscientiously and give to them the best that is in you, without regard for yourself either physically or financially. It is then, I repeat, that you have come upon success and have justified the title which is to be bestowed upon you tomorrow: *Physician.*

At this point it would appear that in spite of my earlier statement about the giving of advice a certain amount has crept into these remarks. For this I apologize, and in extenuation and in conclusion, may I summarize with this charge to each one of you:

"So live that when thy summons comes to join that innumerable caravan"... it may be said of you in the words of Robert Burns:

"An honest man lies here at rest,
As e'er God, with his image blest;
The friend of man, the friend of truth,
The friend of age, the guide of youth:
Few hearts like his, with virtue warm'd
Few heads with knowledge so informed,
If there's another world, he lives in bliss
If there is none, he made the best of this."

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ONE HUNDRED AND TWELFTH PROGRAM MEETING

The One Hundred and Twelfth Program Meeting was held on Tuesday, March 25, 1941 in Gordon Wilson Memorial Hall. The following program was presented:

"The Influence of Tobacco Mosaic Virus on Cellular Respiration," by Drs. M. W. Woods and H. G. duBuy, Department of Botany, Division of Plant Pathology and Physiology, University of Maryland, College Park.

"Testosterone Propionate and Body Growth," by Drs. H. S. Rubinstein and M. L. Solomon, Laboratory for Neuro-Endocrine Research, Surgical Division, Sinai Hospital, Baltimore.

Abstracts of these papers follow.

THE INFLUENCE OF TOBACCO MOSAIC VIRUS ON CELLULAR RESPIRATION*†

M. W. Woods, Ph.D. and H. G. du Buy, Ph.D.

COLLEGE PARK, MD.

Oxygen respiration in healthy Turkish tobacco leaves is accomplished by both cyanide-sensitive and cyanide-resistant systems of enzymes. Since the energy for protoplasmic streaming is derived from respiratory processes, the *rate* of streaming can be used as an index to the respiration of individual cells. Oxygen respiration was measured directly in a modification of the polarographic respirometer and in a modified Fenn differential volumeter, and indirectly by the rate of protoplasmic streaming. By the use of a half-leaf technic, virus titre and respiratory activity were measured in the same tissue.

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* From the Department of Botany, Division of Plant Pathology and Physiology, University of Maryland, College Park, Md.

† Read at the meeting of the University of Maryland Biological Society, March 25, 1941.

There are two cyanide-sensitive systems which can be separated from each other by means of their "critical oxygen tensions." These have been arbitrarily designated as the A and B systems. The third, or C system, is resistant to cyanide and is able to function at lower oxygen tensions than either the A or B components. Infection with tobacco mosaic virus protein (*Marmor tabaci* Holmes) results in inhibition of the A system within forty-eight to seventy-two hours. The B system is apparently not affected, but the C component is greatly increased in activity *following* inhibition of A.

The effect on respiration apparently occurs only after development of a high concentration of virus. Since it has been found that virus multiplication is reversibly inhibited by cyanide (HCN, NaCN or KCN) in the same way that respiration is affected, the virus synthesis must be dependent on the activity of this cyanide-sensitive respiratory system. The virus may thus prevent its own further multiplication by bringing about the inhibition of this respiratory mechanism.

The results of this study indicate the importance of specific respiratory processes of the cell in relation to virus synthesis.

TESTOSTERONE PROPIONATE AND BODY GROWTH*†

H. S. Rubinstein, M. D., PhD. and M. L. Solomon, B. S., M.D.

BALTIMORE, MD.

Castration in the male rat results in growth depression. It was therefore believed that the sex hormone would stimulate body growth. When such animals were treated with one mg. testosterone propionate daily, however, it was observed that the growth of the normal animals was inhibited to an almost equal degree as that observed following castration. The depression resulting from both castration and the administration of testosterone propionate appeared paradoxical. When it was noted that the normal animals who had received testosterone propionate possessed testes which had been greatly inhibited in their growth, it was thought that testosterone propionate through inhibition of the testis had produced an experimental castration effect, thus leading to the inhibited body growth similar to that observed after actual castration.

This same dosage of testosterone propionate was accordingly administered to castrated rats in order to test whether this hormone would make such castrates grow. Such animals, however, showed a still greater depression than either the untreated castrates or the treated normals. It appeared as if the growth depressing effect of the testosterone propionate had been added to the growth depressing effect of castration.

Because of the growth depression following castration and because others (e.g., Zondek) had observed typical pictures of pituitary dwarfism following the excessive use of estrogenic substances, it was felt that the doses of androgens used in the first series of experiments were likewise excessive.

Normal animals were then injected with a dose of 50 gamma testosterone propionate daily for twenty-six to eighty days. It was noted that their body growth became significantly greater than that of the controls. Similar experiments conducted upon castrated rats (using similar doses) disclosed that the treated castrates now became heavier instead of lighter than their castrate brother controls, to a degree which was found to be statistically "probably significant."

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* From the Laboratory for Neuro-Endocrine Research, Surgical Division, Sinai Hospital, Baltimore, Md.

† Read at the meeting of the University of Maryland Biological Society, March 25, 1941.

In another series of experiments, again using the smaller dose (50 gamma) of testosterone propionate, it was observed that normal treated animals became significantly heavier than normal untreated controls, that castrated untreated rats became significantly lighter than castrated treated animals, and that treated castrated rats displayed growth curves which were practically identical with those of normal untreated litter mate brothers.

It should be stressed, however, that care must be exercised in carrying out such experiments on growth since it has been shown that similarly aged animals, similarly treated, may respond differently depending upon the season of the year during which the observations on growth are made. Groups observed beginning October, January and July (from ages twenty-six to eighty days) were found to gain significantly on 50 gamma daily; those observed from April through May and June failed to grow beyond that of their controls.

While one mg. testosterone propionate administered daily inhibits growth by what is believed to result from pituitary depression and 50 gamma stimulates growth sufficiently to restore the castrate's growth curve to that of the normal, it is not yet felt that 50 gamma of testosterone propionate is the ideal dose for replacement in the castrated rat. This is based on observations made in the pituitary glands of these castrated treated animals in which it was noted that castration cells, in spite of treatment, still persist.

ONE HUNDRED AND THIRTEENTH PROGRAM MEETING

The One Hundred and Thirteenth Program Meeting was held on Tuesday, April 29, 1941 in Gordon Wilson Memorial Hall. The following papers were read:

"An Experimental Study of the Effect of the Pituitary Gland on the Digestive Tract and Carbohydrate Metabolism. A Preliminary Report," by Drs. Samuel Morrison and Maurice Feldman, Department of Gastro-Enterology, School of Medicine, University of Maryland.

"The Relationship of Vitamin C to Sulfanilamide Action," by Mr. Leonard Karel, Department of Pharmacology, School of Pharmacy, University of Maryland.

Abstracts of these papers follow.

AN EXPERIMENTAL STUDY OF THE EFFECT OF THE PITUITARY GLAND ON THE DIGESTIVE TRACT AND CARBOHYDRATE METABOLISM. A PRELIMINARY REPORT*†

Samuel Morrison, M.D. and Maurice Feldman, M.D.

BALTIMORE, MD.

In an attempt to answer the question, is the pituitary body essential for the functioning of the digestive tract, the following studies were made:

1. The effect of anterior lobe pituitary extract on normal gastro-intestinal motility.
2. The effect of hypophysectomy upon the motility of the digestive tract.
3. The effect of anterior lobe pituitary extract on the digestive tract of the hypophysectomized animals.

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* From the Department of Gastro-Enterology, School of Medicine, University of Maryland.

† Read at the meeting of the University of Maryland Biological Society, April 29, 1941.

4. The interrelationship between the thyroid and the pituitary body and their effect upon the digestive tract.
5. The relationship between the pituitary and thyroid glands and gastric secretion.

Comparative sugar absorption studies in the thyroidectomized and hypophysectomized animal were also made.

THE RELATIONSHIP OF VITAMIN C TO SULFANILAMIDE ACTION*†

Leonard Karel

BALTIMORE, MD.

A single random determination of the ascorbic acid content of the plasma of guinea pigs is not indicative of the state of vitamin C saturation of the tissues of the animal One must know not only the vitamin C intake of the animal, but also the time elapsing between the last intake of the vitamin and the withdrawal of the blood sample.

Both the oxidized and the reduced forms of ascorbic acid interfere with the determination of free sulfanilamide by the method of Bratton and Marshall when the ratio of ascorbic acid to sulfanilamide is 1:2 or greater.

The heating of ascorbic acid and sulfanilamide together on a water bath for one hour interferes with the recovery of sulfanilamide even when the ratio of ascorbic acid to sulfanilamide is as low as 1:20.

Vitamin C has no influence on the acute toxicity of sulfanilamide for guinea pigs. The LD_{50} for fasting guinea pigs (2.16 grams per kilogram body weight) is significantly different from that for nonfasting guinea pigs (3.13).

Sulfanilamide is effective in the treatment of group C streptococci infections in guinea pigs, but neither high ascorbic acid content nor depletion of vitamin C for ten days influences the effect of sulfanilamide.

In the absence of sulfanilamide treatment, the course of the infection as measured by survival time is not influenced by either high ascorbic acid content or by ten days of vitamin C depletion.

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* From the Department of Pharmacology, School of Pharmacy, University of Maryland.

† Read at the meeting of the Maryland Biological Society, April 29, 1941.

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The names listed above are officers for the term beginning July 1, 1941 and ending June 30, 1942

PRE-COMMENCEMENT EXERCISES

JUNE 6, 1941

The Lyric, Baltimore

ORDER OF EXERCISES

I. The Procession: *The Coronation March*, Meyerbeer; *Pomp and Circumstance*, Elgar.

II. Invocation: Rev. Boyd R. Howarth, Rector of the Memorial Protestant Episcopal Church.

III. Greeting: H. C. Byrd, B.S., LL.D., D.Sc., *President, University of Maryland.*

IV. The Address: Louis H. Douglass, M.D., *Professor of Obstetrics, School of Medicine.*

V. Introduction of Medical Council: H. Boyd Wylie, M.D., *Acting Dean, School of Medicine.*

Arthur M. Shipley, M.D., D.Sc., *Professor of Surgery*
Hugh R. Spencer, M.D., *Professor of Pathology*
H. Boyd Wylie, M.D., *Acting Dean of the Faculty and Professor of Biochemistry*
Carl L. Davis, M.D., *Professor of Anatomy*
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William R. Amberson, Ph.D., *Professor of Physiology*
Louis H. Douglass, M.D., *Professor of Obstetrics*

VI. Introduction of Graduates: John E. Savage, M.D., *Associate in Obstetrics and Assistant in Pathology.*

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Norval Foard Kemp
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George John Ulrich
Lester Aubrey Wall, Jr.
Raymond Kief Thompson

The Dr. A. Bradley Gaither Memorial Prize of $25.00 for the best work in genito-urinary surgery during the senior year: Raymond N. Malouf.

VIII. ADMINISTERING OF HIPPOCRATIC OATH: MAURICE C. PINCOFFS, M.D., *Professor of Medicine.*

IX. BENEDICTION: REV. BOYD R. HOWARTH, Rector of the Memorial Protestant Episcopal Church.

X. THE RECESSION: *March of the Priests*, Mendelssohn.

THE MARSHALLS

Chief Marshall, HARRY C. HULL, M.D.

JOHN C. DUMLER, B.S., M.D.
THURSTON R. ADAMS, M.D.
HENRY F. ULLRICH, D.SC., M.D.
GEORGE H. YEAGER, B.S., M.D.

COMMENCEMENT EXERCISES

JUNE 7, 1941

Ritchie Coliseum, College Park

COMMENCEMENT PROGRAM

CONCERT—MARCH—*The Stars and Stripes Forever*—Sousa.
OVERTURE—*The New Moon*—Romberg.
SUITE IN FOUR PARTS—*Atlantis* (*The Lost Continent*)—Safranek.
Nocturne and Morning Hymn of Praise
A Court Function

I Love Thee (The Prince And Aana)
The Destruction of Atlantis
WALTZ—*Return of Spring*—Waldteufel.
SELECTION—*Jack O'Lantern*—Caryll.
INTERMEZZO—*In a Monastery Garden*—Ketelby. (The University of Maryland Band.)

OTTO SIEBENEICHEN, *Director*

THE ACADEMIC PROCESSION—PROCESSIONAL MARCH—*Aida*—Verdi.
INVOCATION—REVEREND JOHN J. MURPHY, St. Cecilia's Church, Baltimore, Maryland.
MUSIC—*My Maryland*—Audience.
ADDRESS—"No Room for Cloisters," Paul V. McNutt, Administrator, Federal Security Agency.
MUSIC—*America*—Audience.
CONFERRING OF HONORARY DEGREES, HONORARY CERTIFICATES, AND DEGREES IN COURSE—President H. C. Byrd.
MUSIC—*The Star Spangled Banner*—Audience.
BENEDICTION—REVEREND JOHN J. MURPHY.
THE ACADEMIC RECESSION—RECESSIONAL MARCH—*National Emblem*—Bagley.

REUNIONS

CLASS OF 1931 OF THE SCHOOL OF MEDICINE, UNIVERSITY OF MARYLAND

The following members of the Class of 1931 were present for their tenth reunion held in conjunction with the annual banquet of the Medical Alumni Association on June 5, 1941 at the Lord Baltimore Hotel:

Drs. Eugene I. Baumgartner, Arthur T. Brice, Melvin B. Davis, Harry Eckstein, John Wesley Edel, Jr., Samuel Feldman, Raymond F. Helfrich, Mark Hollander, Kent M. Hornbrook, Walter J. Keefe, Harry V. Langeluttig, Paul Lubin, W. B. Moyers, Manuel Rodriguez Ema, Emanuel A. Schimunek, William M. Seabold, Albert J. Shochat, Arthur G. Siwinski, Solomon Smith, and Robert B. Taylor.

Dr. Raymond F. Helfrich was in charge of arrangements for the reunion.

CLASS OF 1916, COLLEGE OF PHYSICIANS AND SURGEONS

Six members of the Class of 1916, College of Physicians and Surgeons, were present at the luncheon and annual meeting of the Medical Alumni Association which was held on June 5, 1941 in the University Hospital. They later attended the Annual Banquet of the Association at the Lord

Baltimore Hotel. In addition to Dr. George McLean of Baltimore, who was in charge of arrangements, the following members of this Class were present:

Drs. Foster A. Beck, Allentown, Pa.; Lucien R. Chaput, Haverhill, Mass.; Wilbur F. Shirkey, Jr., Charleston, W. Va.; Henry F. Buettner and Maurice Feldman of Baltimore.

After the banquet the members met for a final get-together.

CLASS OF 1916, SCHOOL OF MEDICINE, UNIVERSITY OF MARYLAND

The Class of 1916 held its twenty-fifth reunion on June 5 and 6, 1941. Festivities started on the afternoon of June 5 with a cocktail party at the Lord Baltimore Hotel. At night twenty-two members of the Class attended the banquet of the Medical Alumni Association. The following afternoon the Class went on a boat cruise and at night a stag dinner was held at the Lord Baltimore Hotel, with Dr. Irving J. Spear as guest of honor. Upon a motion by Dr. Frank C. Marino a committee was appointed to complete arrangements for a scholarship endowment at the University of Maryland in memory of the deceased members of the Class of 1916. Dr. Frank C. Marino was elected president of the class and Dr. Joseph J. Roberts, secretary. Those who attended this dinner were:

Drs. Franklin B. Anderson, George A. Bawden, E. H. Benson, Howell I. Hammer, Benjamin M. Jaffe, Frank C. Marino, Joseph J. Roberts, F. Frederick Ruzicka, Samuel Snyder, of Baltimore; A. W. Reier, Dundalk, Md.; Edward P. Thomas, Frederick, Md.; William F. Williams, Cumberland, Md.; Bruce Brumbaugh, Elkridge, Md.; Everett L. Bishop, Atlanta, Ga.; E. N. Boccanegra Lopez and Michael Cavallo, New York City; Thomas L. Bray, Plymouth, N. C.; Charles R. Brooke, Newark, N. J.; H. F. Carrasquillo, Utuado, Puerto Rico; William J. Dillon, Springfield, Mass.; John E. Evans, Wilmington, N. C.; William T. Ferneyhough, Reidsville, N. C.; Clarence P. L. Gannon, Kingston, N. Y.; Manuel Garcia de Quevedo, Anasco, Puerto Rico; Bowers H. Growt, Addison, Mich.; Lewis W. Glatzau, Daytona Beach, Fla.; Robert H. Noell, Rocky Mount, N. C.; Vincent J. Oddo, Providence, R. I.; Harold M. Stein, Paterson, N. J.; Norwood W. Voss, Wilmington, Del.; Maurice C. Wentz, York, Pa.

Through the kindness of Mrs. F. F. Ruzicka the visiting ladies were taken on trips to Washington and Annapolis and entertained at Mrs. Ruzicka's home for dinner. The committee in charge of the reunion feels certain that the entertainment provided was enjoyed thoroughly, and that the members of the Class look forward to meeting again in the near future.

CLASS OF 1911 OF THE SCHOOL OF MEDICINE, UNIVERSITY OF MARYLAND

Under the chairmanship of Dr. Walter C. Bacon, the Class of 1911 held its thirtieth annual reunion on June 5 and 6, 1941. Those present were:

Drs. Sydney Wallenstein, Louis Douglass, Ralph Vreeland, Jacob Greengrass, Charles H. Keesor, H. A. Codington, Ernest Bullock, A. E. Brown, Raymond Hussey, Dallas C.

Speas, Henry Athey, Kenneth B. Jones, E. E. Nichols, Walter S. Niblett, and Walter C. Bacon.

CLASS OF 1911 OF THE COLLEGE OF PHYSICIANS AND SURGEONS

Nine members of the Class of 1911 of the College of Physicians and Surgeons attended the thirtieth annual reunion on June 5 and 6, 1941. Dr. John F. Hogan was in charge of arrangements. The following were present:

Drs. David Deutschman, B. H. Swint, John Shea, Joseph Kilbourn, William T. Gocke, A. F. Lawson, Caldwell Woodruff, Frank L. Jennings, and John F. Hogan.

CLASS OF 1906 OF THE SCHOOL OF MEDICINE, UNIVERSITY OF MARYLAND

The following members attended the thirty-fifth reunion of the Class of 1906:

Drs. N. W. Hershner, E. W. White, Ralph Dees, J. M. Jarrell, and Harry A. Cantwell, chairman.

CLASS OF 1901 OF THE COLLEGE OF PHYSICIANS AND SURGEONS

The fortieth annual reunion of the Class of 1901 of the College of Physicians and Surgeons was held on June 5 and 6, 1941. Dr. Henry R. McGraw was chairman for the occasion. Those present were:

Drs. S. D. Sutliff, L. J. Rosenthal, A. B. Vanderbeek, F. T. Marr, Tunis Nunemaker, Samuel Weinberg, and Henry R. McGraw.

CLASS OF 1901 OF THE SCHOOL OF MEDICINE, UNIVERSITY OF MARYLAND

Dr. J. Dawson Reeder was chairman for the fortieth annual reunion of the Class of 1901. Those who attended were:

Rear Admiral Benjamin H. Dorsey, Albert S. Harden, A. F. Williams, Jr., W. T. Messmore, Norman S. Dudley, Robert Glass, and J. Dawson Reeder.

CLASS OF 1896 OF THE COLLEGE OF PHYSICIANS AND SURGEONS

Four members were present at the forty-fifth annual reunion of the Class of 1896 of the College of Physicians and Surgeons:

Drs. Hugh F. Cook, Harvey G. Beck, Jesse Coggins, and Albertus Cotton, chairman.

CLASS OF 1886 OF THE SCHOOL OF MEDICINE, UNIVERSITY OF MARYLAND

The following members attended the fifty-fifth annual reunion of the Class of 1886:

Drs. Harry Friedenwald, C. W. Pritchett, R. Sumter Griffith, M. Gibson Porter, and William P. E. Wyse.

THE 134TH ANNUAL BANQUET

The 134th Annual Banquet of the Medical Alumni Association of the University of Maryland was held on June 5, 1941 at the Lord Baltimore Hotel, Baltimore. The speakers on the program were as follows:

Dr. H. C. Byrd, President of the University of Maryland
Mr. E. P. Coffey, Guest Speaker
Dr. William H. Pearce, President of the Baltimore City Medical Society, Guest Speaker
John E. Esnard, President of the Senior Class
Dr. H. Boyd Wylie, Acting Dean of the School of Medicine
Dr. Charles Bagley, Jr., Toastmaster

In addition to the speakers, the following were guests of honor:
President H. C. Byrd
His Excellency Herbert R. O'Conor, Governor of Maryland
The Honorable Howard W. Jackson, Mayor of Baltimore
Members of the Board of Regents
Graduates of the Class of 1886
Graduates of the Class of 1941

REGISTRANTS FOR SPRING ACTIVITIES

The following alumni registered during the Spring Activities:

Drs. Lewis W. Glatzau, Daytona Beach, Fla.; A. B. Vanderbeek, Paterson, N. J.; Frank McDede, Paterson, N. J.; Frank T. Marr, Chillicothe, Ohio; V. G. Williams, Grantville, Ga.; S. T. Lowry, San Antonio, Texas; D. C. Speas, Winston-Salem, N. C.; Stanley W. Matthews, Fort Jackson, S. C.; M. Rodriguez-Ema, Santurce, P. R.; B. H. Swint, Charleston, W. Va.; A. S. Harden, Newark, N. J.; Hugh F. Cook, Newark, N. J.; M. G. de Quevedo, Anasco, P. R.; Charles H. Keesor, Wheeling, W. Va.; David Sashin, New York City; Abel Gordon, Passaic, N. J.; Daniel Helfond, Glendale, L. I.; Irving Bronstein, Yonkers, N. Y.; Frederick Sabin, Little Falls, N. Y.; Harold H. Freedman, Freehold, N. J.; R. Sumter Griffith, Waynesboro, Va.; Ralph Dees, Greensboro, N. C.; Mortimer H. Williams, Roanoke, Va.; Guy L. Whicker, Kannapolis, N. C.; Everett L. Bishop, Atlanta, Ga.; Joseph B. Kilbourn, Hartford, Conn.; W. T. Gocke, Clarksburg, W. Va.; John Shea, Bridgeport, Conn.; Elizabeth B. Sherman, Front Royal, Va.; H. B. McDonnell, College Park, Md.; J. A. Krosnoff, Bentleyville, Pa.; Herbert A. Codington, J. E. Evans and Ernest S. Bullock, Wilmington, N. C.; K. M. Jarrell, Beckley, W. Va.; A. F. Williams, Wilson, N. C.; S. D. Pentecoste, Irvington, N. J.; S. D. Sutliff, Shippensburg, Pa.; E. Irving Baumgartner, Oakland, Md.; Tunis Nunemaker, Portsmouth, Ohio; Bruce Barnes, Seaford, Del.; W. T. Messmore, Uniontown, Pa.;

W. J. Dillon, Springfield, Mass.; C. P. Gannon, Kingston, N. Y.; A. F. Lawson, Weston, W. Va.; J. Henry Orff, Shillington, Pa.; V. J. Oddo, Providence, R. I.; O. C. Moulton, Reno, Nev.; C. W. Pritchett, Danville, Va.; Samuel Feldman, New York City; Lucien R. Chaput, Haverhill, Mass.; J. S. Grabill, Mt. Airy, Md.; Arley V. McCoy, Wheeling, W. Va.; Richardson Joyner, Suffolk, Va.; A. W. Valentine, Washington, D. C.; Norman S. Dudley, Church Hill, Md.; John Reynolds, Akron, Ohio; I. Terr, Woodmere, N. Y.; H. Eckstein, Staten Island, N. Y.; Harry A. Cantwell, North East, Md.; F. H. Jaklitsch, Brooklyn, N. Y.; George H. Steuart, Ottoman, Va.; K. M. Hornbrook, New Martinsville, W. Va.; E. W. White, Poolesvilles, Md.; Ralph Vreeland, Paterson, N. J.; J. J. Greengrass, Paterson, N. J.; Foster Beck, Allentown, Pa.; Henry R. McGraw, Denver, Colo.; J. W. Schilling, Erie, Pa.; A. E. Brown, Greenville, S. C.; L. J. Weseley, Brooklyn, N. Y.; J. Weinstein, Brooklyn, N. Y. Drs. Margaret B. Ballard, E. A. Smith, G. J. Wells, A. G. Siwinski, M. L. Solomon, Edgar B. Friedenwald, H. Boyd Wylie, Harry M. Robinson, Jr., Carl F. Benson, F. T. Rogers, N. R. Kleiman, Arthur Hebb, Clewell Howell, Lewis P. Gundry, A. H. Finkelstein, H. B. Athey, Albertus Cotton, John Evans, E. C. Reitzel, Herbert E. Zepp, H. I. Hammer, J. E. Savage, Ralph P. Truitt, Louis H. Douglass, L. C. Dobihal, George McLean, J. Morley Hoag, Samuel Weinberg, C. Loring Joslin, M. B. Davis, Mark Hollander, Monte Edwards, William H. Pearce, Walter C. Bacon, J. M. H. Rowland, and Philibert Artigiani of Baltimore.

ITEMS

THE DR. WILLIAM RAYMOND SANDERSON BEQUEST OF $1,000

The School of Medicine has received a check in the amount of $1,000 from the estate of Dr. William Raymond Sanderson, University of Maryland, 1882, in conformity to the following bequest:

"I give and bequeath to my Alma Mater, the University of Maryland, School of Medicine, as a token of my esteem for said school, the sum of One Thousand Dollars ($1,000.00), said money to be used for such purpose as the faculty of said School may see fit."

SCHOLARSHIP SUPPORT—AN INNOVATION

A promise from each student of the 1941 graduating class to give one dollar on graduation and one dollar a year hereafter, in support of alumni scholarship aid to needy medical students, was announced by the class president, John E. Esnard, at the Alumni banquet on June 5, 1941. In fulfillment of the promise ninety-two members deposited $126.00 with the treasurer of the Medical Alumni Association.

This wholesome gesture was the subject of many favorable comments by groups of older alumni at the banquet. In addition, it was announced by Dr. Charles Bagley, Jr., the outgoing president of the Medical Alumni Association, that the younger classes in the School of Medicine were planning to develop the action of the Class of 1941 into a tradition.

Mrs. Ruth Lee Briscoe, Librarian of the School of Medicine, presented a paper at the March meeting of the Washington-Custis Chapter, Maryland Society Daughters of the American Revolution. The title of the paper was "The Visit of General LaFayette to the University of Maryland." This was followed by a description of the General LaFayette flag presented to the Library of the School of Medicine on December 14, 1917 by the Maryland Society Daughters of the American Revolution.

Dr. Grant E. Ward, Associate Professor of Surgery, delivered an illustrated lecture on "The Importance of Early Recognition of Cancer" at the meeting of the Maryland Academy of Medicine and Surgery which was held on April 15, 1941. On the same program a report from the Legislative Committee was read by Dr. Thomas S. Bowyer, Instructor in Gynecology. Dr. Thomas P. Sprunt, Professor of Clinical Medicine, spoke on "Pneumonia."

Christopher C. Shaw, (U. of Md. 1931) Lieutenant Commander, M.C.,-V.(S.) U.S.N.R., has been called to active duty at the Chelsea Naval Hospital. Lieut. Comdr. Shaw became a fellow of the American College of Physicians on April 23, 1941.

Dr. Herman Chor (U. of Md. 1928), instructor in the department of nervous and mental diseases at Northwestern University for seven years, will be guest lecturer in his field at Leland Stanford University during the summer months as a contributor to the school's fiftieth anniversary celebration.

MEDICAL LIBRARY NOTES

Dr. John E. Savage, Acting Superintendent of the University Hospital, Associate in Obstetrics and Assistant in Pathology, was appointed to the Library Committee of the School of Medicine in the fall of 1940. Shortly afterward Dr. Savage became a supporting member of the Medical Library Association. Particularly interested in rare and historical material, Dr. Savage has long been collecting old books on the subject of obstetrics.

Dr. Louis A. M. Krause, Associate Professor of Medicine, gave a weekly series of lectures on the history of medicine during March and April, 1941. These were held in the Bressler Building.

Miss Julia E. Wilson, of the Medical Library staff, attended the annual meeting of the American Association of the History of Medicine held in Atlantic City from May 4 to May 6. The paper "Early Nineteenth Century Books on Heart Disease" read at this meeting was of particular interest from the bibliographical viewpoint.

Recent gifts to the Medical Library have included over twenty-five volumes from the office of Dr. Maurice Pincoffs, Professor of Medicine and Editor of Annals of Internal Medicine.

The mailing list of the Bulletin of the School of Medicine has been revised with the approval of the Acting Dean, Dr. H. B. Wylie, so that most of the medical libraries in the Latin American countries are now receiving the Bulletin regularly. The Medical Library benefits considerably thereby as many foreign journals are received in exchange.

The University Librarian wishes to extend his thanks to all who cooperated in submitting material for the exhibit "Baltimore Educators" held recently at the Enoch Pratt Free Library. The members of the faculty of the School of Medicine were well represented as fifty-nine out of a total of 248 items submitted by the University as a whole came from their pens. In the judgment of officials of the Enoch Pratt Free Library the exhibit was so successful that it has been decided to hold another in 1942 covering publications of 1941. An invitation for the University of Maryland to participate has already been extended and accepted.

Carl W. E. Hintz, A.B., A.M.L.S.,
University Librarian.

UNIVERSITY OF MARYLAND
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SCHOOL OF MEDICINE

Dr. Fred Wharton Rankin

DR. FRED WHARTON RANKIN—PRESIDENT-ELECT OF THE AMERICAN MEDICAL ASSOCIATION

The House of Delegates of the American Medical Association has elected a graduate of the School of Medicine of the University of Maryland to the highest honor in the province of scientific medicine. Dr. Rankin has participated notably in the affairs of the American Medical Association and of many other scientific bodies. He was born in Mooresville, North Carolina on December 20, 1886. After receiving his bachelor of arts degree from Davidson College in 1905 he received his degree of doctor of medicine from the University of Maryland in 1909 and the degree of master of arts from St. John's College in 1913. He was also made honorable doctor of sciences by Davidson College, his alma mater, in 1937, and honorary doctor of laws by the University of Maryland in 1939.

Following his graduation in medicine Dr. Rankin became a resident surgeon at the University Hospital from 1909 to 1912, and served as assistant demonstrator of anatomy and associate in surgery at the School of Medicine of the University of Maryland from 1913 to 1916. He then joined the Mayo Clinic in Rochester, Minnesota, acting as assistant surgeon at St. Mary's Hospital from 1916 to 1923. He was professor of surgery at the University of Louisville, 1922–23, and served as surgeon to the Mayo Clinic and as associate professor at the University of Minnesota Medical School, Mayo Foundation, from 1926 to 1933. He then removed to Lexington, Kentucky, where he became surgeon to St. Joseph's and the Good Samaritan Hospitals, with which he has been associated since January 1, 1934. Dr. Rankin also is Clinical Professor of Surgery at the University of Louisville. In the World War he served as a major in the Medical Corps for seventeen months and was attached to the First Army Corps, 4th and 26th divisions in France, as commanding officer of Base Hospital No. 26. He is now a colonel in the Medical Reserve Corps.

Dr. Rankin has been honored by many medical organizations, including the presidency of the Southern Surgical Association and the Southeastern Surgical Congress. He is a fellow of the American College of Surgeons, and a member of the American Surgical Association, American Proctologic Society, Eastern and Western Surgical Associations, Southern Medical Association, and many surgical clubs and medical fraternities. He was one of the founder members of the American Board of Surgery, representing in that body the Section on Surgery of the American Medical Association. His contributions to medical literature include a monograph on "Surgery of the Colon"; a work on "The Colon, Rectum and Anus," published in 1932 jointly with Drs. J. A. Bargen and L. A. Buie, and a work on "Cancer of the Colon and Rectum," published with Dr. A. S. Graham in 1939.

He has also contributed chapters on surgery of the colon, with particular reference to carcinoma, in several systems of surgery.

In the American Medical Association Dr. Rankin has been especially active. He was a member of the House of Delegates and represented the Section on Surgery from 1935 through 1940. On several occasions he has aided in the work of the reference committees. In 1936 he was appointed to the membership of the Council on Medical Education and Hospitals, and he has been active in the work of that body. He was appointed by the speaker of the House of Delegates a member of the Committee on Medical Preparedness in 1940 and has been assiduous in the duties of that committee and especially in military preparedness in the Fifth Corps Area. In 1923 Dr. Rankin married Miss Edith Mayo, a daughter of Dr. Charles H. Mayo, and he has four children. Almost immediately following his election he was deluged with invitations to attend many meetings of state and other medical organizations, indicating not only his popularity with the medical profession but his devotion to the work of organized medicine.

THE BRESSLER ALUMNI RESEARCH FUND

University of Maryland School of Medicine, Baltimore, Maryland

The following is a brief resumé of the research activities which have been made possible by grants-in-aid from the Bressler Alumni Research Fund. This represents the manner in which the funds contributed by subscription are being used to advance scientific knowledge in medicine. Further information will gladly be given upon request to Dr. John C. Krantz, Jr., Secretary, Bressler Alumni Research Fund, University of Maryland, School of Medicine, Baltimore, Maryland.

Dr. Glenn H. Algire assisted by Miss Mellisa Pyle: Certain characteristic features of the skin in thyrotoxicosis have been noted in the medical clinic as a part of a study aimed at differentiating normal from thyrotoxic integument. This study concerns the chemical characteristics of the skin of such thyrotoxic humans and animals.

A technic has been elaborated for the determination of cholesterol in the skin, and a comparison is being made of the skin in normal and thyrotoxic humans, salamanders and cats.

Dr. William R. Amberson: A method of preparing dried human and animal hemoglobin is being developed in the hope that this dried material may take the place of dried plasma in infusion and transfusion work. The lyophil process of Mudd and Flosdorf has proved adequate for the drying, but it has not yet been found possible to prepare hemoglobin which, on resolution, gives 100 per cent oxyhemoglobin. The physical and chemical conditions necessary to prevent the formation of methemoglobin during the drying process are being studied. To date the best samples are about two-thirds active. As soon as a sufficient quantity of good dried material is available, animal experiments will be made to test the adequacy of the solutions for replacing lost blood.

Dr. E. G. Boettiger in collaboration with Dr. D. C. Smith and Dr. J. M. Hundley, Jr.: The analysis of ovarian function by quantitative determination of urinary estrogens is only possible when the effects which various organs have on these substances are taken into account. One of the organs intimately concerned is the kidney. This study treats of the effects on the kidney of estrogens brought to it by the blood. Inulin and diodrast clearances will be studied in the dog, relating them to estrogen clearances. Later this work will be continued on the human. The inulin method has been worked out satisfactorily, and details on a new diodrast method will soon be available for use. A large rat colony has been started for bio-assay of the estrogens. At present experiments are being conducted with a chemical method of estrogen determination to supplement the bio-assay.

Professor O. G. Harne: A colony of Macacus rhesus monkeys has been established and the necessary research apparatus for the study of the life duration of the red blood cell in primate animals has been purchased. The experiments follow closely the same line of procedure that was used in original work on rats conducted by the university. It has already been determined that the monkey reacts favorably to hemorrhage. Complete blood studies are in full progress. The indication at present is that the red blood cell in the primate is long lived. The experiments, however, are as yet too incomplete to allow of a definite statement at this time.

Dr. John C. Krantz, Jr.: A method has been developed to determine nitrites in blood. It has been shown that nitrites occur naturally in the normal blood of the dog, the Macacus rhesus monkey, the cow and the human being. The concentration seems to be of same order of magnitude, namely, about 1 to 100,000,000. Plans are being made to study the significance of the presence of nitrites in the blood.

Dr. Robert H. Oster and Dr. James E. P. Toman: A vacuum tube amplifier, cathode ray tube, and inkwriter have been adapted for use with a screened subject cage. With this apparatus a series of electro-encephalograms have been obtained on normal human subjects. A series of brain wave recordings are being made with the improved apparatus on a number of abnormal subjects, including cases of epilepsy and pyromania, as an aid toward a better understanding of the neurologic basis of the abnormalities.

Dr. Dietrich C. Smith: In collaboration with Dr. James C. Lipsett, Weaver Fellow in the Department of Physiology, the metabolic rates of two species of Amblystoma are being determined. *A. tigrinum* is a common American salamander which shows normal metamorphosis from an aquatic larval form to the terrestrial adult. *A. mexicanum* (axolotl), found in the lakes around Mexico City, does not usually metamorphose, but remains in an infantile condition in the water. The study is directed to the discovery of differences in oxygen consumption between the two species, which may correlate with their different rates and extents of glandular development. Hybrid animals, produced by crossing the two species, are also being studied.

Mr. LeRoy W. Tilt, Jr.: The histologic structure of the lung alveolus of the rabbit is being investigated by studying successive fetal stages of a given lung area. Models of the twenty-six day stage have been made and work is now in progress on similar models of the earlier stages of development. In making a three dimensional interpretation of the individual serial sections from which the models were made, it is hoped to piece together a complete story of the development of the alveolar wall which at the present time is a matter of dispute.

Dr. Milton Solomon in conjunction with Dr. Eduard Uhlenhuth: The object of this investigation is to ascertain whether the hypophysis may be activated by certain substances through their introduction into the brain. A technic has been elaborated for the introduction of such substances into the ventricles of the brain without serious disturbance to the well-being of the animal. It is, however, too early to predict whether this procedure will yield the desired results. Experiments to determine this are in progress.

Dr. George H. Yeager: Immediate investigative work has consisted of attempting to evaluate the various technics used in appendicectomy. Phases being considered are: invagination of the stump versus noninvagination; silk versus catgut. Pathologic studies, in regard to peritoneal reaction, are being made in its relationship to this problem with the cooperation of the Department of Pathology.

In addition, some preliminary work is being done with the purpose of possibly studying splenomegaly in its relationship to Banti's disease.

All members of the Alumni Association and friends of the School of Medicine, University of Maryland, who did not contribute to this research fund and wish to do so are cordially invited to send in their contributions after executing the following blank. This money is making it possible for the faculty and research students of the School of Medicine to carry on medical research in the new Bressler Building and other departments of the School. It is hoped that there will be a generous response to this invitation.

UNIVERSITY OF MARYLAND
SCHOOL OF MEDICINE
THE BRESSLER ALUMNI RESEARCH CAMPAIGN

To support research in the Bressler Building of the School of Medicine I pledge

☐ ____ ☐ $25 ☐ $20 ☐ $15 ☐ $10 ☐ $5 ☐ ____

(Please check or insert amount)

per annum for 5 years. My first contribution for this amount is enclosed.

*Dr.*______________________________

MAKE CHECKS PAYABLE TO THE BRESSLER ALUMNI RESEARCH CAMPAIGN.

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Battaglia, Dominick	Baltimore, Md.
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Bawden, George A.	Baltimore, Md.
Bayer, Ira E., Jr.	Baltimore, Md.
Bayley, George	Baltimore, Md.
Baylus, Meyer	Baltimore, Md.
Beck, Frances F.	Baltimore, Md.
Beck, Foster A.	Allentown, Pa.
Beck, Harvey	Baltimore, Md.
Beck, Nathaniel	Baltimore, Md.
Beckner, Wm. F.	Huntington, W. Va.
Bell, Frederick K.	Baltimore, Md.
Benesuns, Joseph	Baltimore, Md.
Benfer, Kenneth L.	York, Pa.
Bennett, George E.	Baltimore, Md.
Benson, Edward H.	Baltimore, Md.
Bercovitz, Nathan	New York City
Berger, Herbert	Staten Island, N. Y.

Berger, William A.........Newark, N. J.
Bernardo, John R...........Bristol, R. I.
Bernstein, Joseph........Baltimore, Md.
Berry, P. E...........Piedmont, W. Va.
Bispham, W. N...........Baltimore, Md.
Black, W. P...........Charleston, W. Va.
Blankenship, Walter......Lancaster, Pa.
Blazejewski, Stanley..Wilkes-Barre, Pa.
Bloom, Lawrence....Phillipsburg, N. J.
Bobbitt, O. H.........Charleston, W. Va.
Bobbitt, Ray M.......Huntington, W. Va.
Boccanegra-Lopez, E. N..New York City
Boettiger, Edward......Baltimore, Md.
Bongiorno, H. D..........Paterson, N. J.
Bordley, James, Jr.......Baltimore, Md.
Bowman, H. D.........Hagerstown, Md.
Bowyer, Thomas S.......Baltimore, Md.
Bradley, J. Edmund......Baltimore, Md.
Brager, Simon..........Baltimore, Md.
Brantigan, O. C.........Baltimore, Md.
Brauer, Selig...........Jersey City, N. J.
Breeding, Earle G.....Washington, D. C.
Breslin, Robert H.......Providence, R. I.
Brice, A. T.............Jefferson, Md.
Bridges, Wm. A...........Towson, Md.
Brill, John L.........Philadelphia, Pa.
Briscoe, Edward..Prince Frederick, Md.
Bronstein, Irving..........Yonkers, N. Y.
Brookfield Pharmacy
Irving Freed and G. D. Kroopnick
Baltimore, Md.
Brown, Leo...........Washington, D. C.
Brown, Paul............Baltimore, Md.
Brumback, Lynn H.....Hagerstown, Md.
Brumbaugh, Benjamin B....Elkridge, Md.
Bubert, Howard M.......Baltimore, Md.
Buettner, Henry F.......Baltimore, Md.
Burgess, Thomas D....Williamson, W. Va.
Burgtorf, George E.....Ellicott City, Md.
Burns, Harold H.........Baltimore, Md.
Burns, Ira............Wilmington, Del.
Burton, Charles H.......Baltimore, Md.
Butts, C. Earl..........Baltimore, Md.
Byerly, M. Paul..........Baltimore, Md.
Byrd, H. C...........College Park, Md.
Byrnes, Harry F........Springfield, Mass.

Cadle, W. R...........Emmitsburg, Md.
Calkins, Irving R.......Springfield, Mass.
Carey, T. Nelson........Baltimore, Md.
Carman, Richard P.....Baltimore, Md.
Carr, C. Jelleff........Baltimore, Md.
Carter, Roy............Baltimore, Md.
Chambers, Earl........Baltimore, Md.
Champe, Preston, Jr...Charleston, W. Va.
Chargin, Louis..........New York City
Chideckel, Maurice......Baltimore, Md.
Chimacoff, Hyman......Newark, N. J.
Clapp, C. A.............Baltimore, Md.
Clark, F. A...........Charleston, W. Va.
Clarke, Carl D..........Baltimore, Md.
Clois, E. E...........Wheeling, W. Va.
Coble, A. C..............Dauphin, Pa.

Cohen, Archie R.......Clear Spring, Md.
Cohen, Herman........Trenton, N. J.
Cohen, M. Marvin........Paterson, N. J.
Cohen, Paul............Salisbury, Md.
Cole, I. D.............Clarksburg, W. Va.
Coll, Thomas L.........Cambridge, Md.
Condry, R. J.............Beckley, W. Va.
Cone, Nellie............Baltimore, Md.
Conway, William S......Springfield, Mass.
Coonan, Thomas J.......Baltimore, Md.
Cooper, B. H...........Glen Lyon, Pa.
Cope, Arthur A.........Hamburg, Pa.
Copeland, Charles E...Charleston, W. Va.
Corbitt, Robert W...Parkersburg, W. Va.
Cotton, Albertus..........Baltimore, Md.
Covington, E. Eugene....Baltimore, Md.
Crisp & Edmunds........Baltimore, Md.
Cronin, D. J.........Huntington, W. Va.
Cross, E. W...........Tarentum, Pa.
Culbreth, David M. R.....Baltimore, Md.
Curry, George R............Reading, Pa.

Dailey, W. Paul........Harrisburg, Pa.
Dailey, Wm. P...........Steelton, Pa.
D'Angelo, Antonio F.......Bristol, R. I.
Daniels, W. F.......Huntington, W. Va.
Davidov, Hyman.........Baltimore, Md.
Davidov, Nathan........Baltimore, Md.
Davies, J. R...........Blossburg, Pa.
Davis, Carl L.........Baltimore, Md.
Davis, Geo. B.....Blue Ridge Summit, Pa.
Davis, H. V.......Chesapeake City, Md.
Day, Lester............Coatesville, Pa.
Deckert, Alan..........Baltimore, Md.
Demarco, S., Jr.........Baltimore, Md.
Demarco, S., Sr.........Baltimore, Md.
Deutschmann, David......New York City
DeVincentis, Henry........Orange, N. J.
Devlin, Joseph A.......New York City
Dickey, Francis G.....Philadelphia, Pa.
Diehl, H. C...........Frostburg, Md.
Diener, Louis..........Baltimore, Md.
Disbrow, Edward.......Worcester, Mass.
Dittmar, Stuart......Chambersburg, Pa.
Dobihal, Louis..........Baltimore, Md.
Dodge, Douglas R.......Pasadena, Calif.
Dolfman, Victor......Philadelphia, Pa.
Donchi, Solomon M.......Newark, N. J.
Donohue, Mildred.......Baltimore, Md.
Dorf, Herman J.........Baltimore, Md.
Dorsey, Benjamin H...Washington, D. C.
Doshay, Lewis J..........New York City
Dougherty, Gerald......Wilmington, Del.
Douglass, Louis H.........Baltimore, Md.
Downes, John R.........Newark, Del.
Downey, Jesse W.........Baltimore, Md.
Dozois, Pierre........Philadelphia, Pa.
Drozd, Joseph..........Baltimore, Md.
Dudley, Norman S.......Church Hill, Md.
Duncan, George A.........Norfolk, Va.
Dunning, H. A. B.......Baltimore, Md.
Dyar, Edna G.........Rockville, Md.

Edwards, Charles Reid	Baltimore, Md.
Edwards, Monte	Baltimore, Md.
Edwards, V. P.	Edwardsville, Pa.
Eliason, H. W.	Cumberland, Md.
Elliott, Alfred	Middleboro, Mass.
Ellis, A. Lee	Baltimore, Md.
Ellis, Fred W.	Baltimore, Md.
England, Welch	Parkersburg, W. Va.
Epstein, H. H.	New York City
Esker, H. H.	Clarksburg, W. Va.
Evans, H. P.	Keystone, W. Va.
Evans, John	Baltimore, Md.
Evans, W. E., Jr.	Baltimore, Md.
Everett, Franklin R.	Dover, Del.
Ewald, August L.	Baltimore, Md.
Fabbri, Remo	Norristown, Pa.
Farr, Robert W.	Chestertown, Md.
Fassett, B. W.	Durham, N. C.
Faw, Wylie M.	Cambridge, Md.
Feder, Aaron	New York City
Feldman, Maurice	Baltimore, Md.
Ferneyhough, Wm. T.	Reedsville, N. C.
Figge, F. M. H.	Baltimore, Md.
Fine, M. James	Newark, N. J.
Finegold, Abraham	Pittsburgh, Pa.
Finegold, Joseph	Pittsburgh, Pa.
Fineman, Jerome	Baltimore, Md.
Finkelstein, A. H.	Baltimore, Md.
Fiscus, J. H.	Greensburg, Pa.
Fisher, C. F.	Clarksburg, W. Va.
Fisher, Charles F.	Salisbury, Md.
Fisher, Harry R.	New York City
Fisher, Joseph O.	New York City
Fisher, R. W.	Morgantown, W. Va.
Fisher, Samuel	Paterson, N. J.
Fisher, W. Henry	Centreville, Md.
Flax, Ira I.	Newark, N. J.
Flippin, Eugene L.	Baltimore, Md.
Flom, Charles	Baltimore, Md.
Forman, Sylvan E.	Baltimore, Md.
Foster, Ruth	New York City
Foxwell, R. K.	Washington, D. C.
France, A. M.	Parkton, Md.
Franklin, Joseph P.	Cumberland, Md.
Fredlock, A. M.	Elkins, W. Va.
Freedom, Leon	Baltimore, Md.
Friedenwald, Edgar B.	Baltimore, Md.
Friedenwald, Harry	Baltimore, Md.
Friedenwald, Julius	Baltimore, Md.
Friedman, Joseph	Westernport, Md.
Friedman, Louis M.	New York City
Friedman, Meyer H.	Trenton, N. J.
Galitz, Philip J.	Brooklyn, N. Y.
Galvin, Thomas K.	Baltimore, Md.
Gann, Mark E.	Baltimore, Md.
Ganz, S. Evans	New York City
Garber, J. S.	Jamaica, N. Y.
Gelb, Jerome	Newark, N. J.
Gellar, Abraham	Brooklyn, N. Y.
Geller, Samuel	Newark, N. J.
Gelman, Sidney	Paterson, N. J.
Gemmill, W. F.	York, Pa.
Gerwig, Walter H., Jr.	Baltimore, Md.
Gillson, Hugh V.	Paterson, N. J.
Ginsburg, Leon	Baltimore, Md.
Gittleman, Sol E.	Brooklyn, N. Y.
Glasgow, Thomas M.	Passaic, N. J.
Glass, Albert	Baltimore, Md.
Glass, Earl F.	Wheeling, W. Va.
Glick, Samuel S.	Baltimore, Md.
Gluckman, Albert G.	Wilmington, Del.
Gocke, Wm. T.	Clarksburg, W. Va.
Godbey, J. R.	Charleston, W. Va.
Goldberg, Ben M.	Trenton, N. J.
Goldberg, Victor	Baltimore, Md.
Goldey, Abert	New York City
Goldman, Alex B.	Brooklyn, N. Y.
Goldman, Bernhard	Pittsburgh, Pa.
Goldsborough, Charles R.	Baltimore, Md.
Goldstein, A. E.	Baltimore, Md.
Goldstein, Milton J.	Scranton, Pa.
Good, B. F.	Lancaster, Pa.
Goodale, Hazel M.	Baltimore, Md.
Goodall, Edwin B.	Boston, Mass.
Goodhand, Chas. L.	Parkersburg, W. Va.
Goodman, Jerome E.	Baltimore, Md.
Gordon, Abel	Passaic, N. J.
Gordon, A. S.	Brooklyn, N. Y.
Gordon, W. C.	Providence, R. I.
Gould, Nathaniel J.	New York City
Gracie, W. A.	Cumberland, Md.
Graff, H. F.	Baltimore, Md.
Greenfeld, Wm.	Baltimore, Md.
Greengold, David B.	Pittsfield, Mass.
Greengrass, J. J.	Paterson, N. J.
Greenwald, Leon	Baltimore, Md.
Greer, Creed C.	Clarksburg, W. Va.
Griffin, M. J.	Fall River, Mass.
Grisinger, G. F.	Charleston, W. Va.
Grossblatt, Philip	Newark, N. J.
Grove, D. B.	Cumberland, Md.
Gundry, Alfred T.	Catonsville, Md.
Gurley, Hubert T.	Baltimore, Md.
Guthrie, J. A.	Huntington, W. Va.
Hachtel, Frank W.	Baltimore, Md.
Hahn, Albert, Jr.	Baltimore, Md.
Hahn, Charles S.	Brooklyn, N. Y.
Haimowitz, Samuel	York, Pa.
Hala, W. W.	Long Island City, N. Y.
Hall, Edward T. Wade	Weston, W. Va.
Halliday, Chas. H.	Baltimore, Md.
Haney, John J.	Trenton, N. J.
Hankin, Samuel	Baltimore, Md.
Hanna, Martin J.	Baltimore, Md.
Harne, O. G.	Baltimore, Md.
Harne, W. G.	Baltimore, Md.
Harper, W. G.	Elkins, W. Va.
Harris, L. N.	Wheeling, W. Va.
Harrison, Charles S.	Wheeling, W. Va.
Hartman, G. O.	Sharon, Pa.
Hartman, I. F.	Buckhannon, W. Va.
Hawkins, A. H.	Cumberland, Md.
Haynes, H. H.	Clarksburg, W. Va.

Heath, F. C.	Weston, W. Va.
Hebb, Arthur	Baltimore, Md.
Hecht, Manes	Baltimore, Md.
Hedges, F. H.	Frederick, Md.
Hedrick, E. H.	Beckley, W. Va.
Hedrick, G. C., Jr.	Glen Ferris, W. Va.
Hedrick, G. C., Sr.	Beckley, W. Va.
Heisley, R. S.	Honesdale, Pa.
Hege, J. Roy	Winston-Salem, N. C.
Helfond, David M.	Brooklyn, N. Y.
Helms, Samuel	Baltimore, Md.
Hendler, H. B.	Lynbrook, N. Y.
Hendler, Manuel	Baltimore, Md.
Herbert, A. N.	Ellicott City, Md.
Hersperger, W. Grafton	Baltimore, Md.
Hertz, Ben	New York City
Hicks, Ira Clay	Huntington, W. Va.
Highstein, Benjamin	Baltimore, Md.
Hildenbrand, Emil J. C.	Wash., D. C.
Himelfarb, Albert	Baltimore, Md.
Hines, Frank	Chestertown, Md.
Hines, Miriam	Chestertown, Md.
Hirsch, Philip	Elmhurst, N. Y.
Hoff, Louis L.	Holyoke, Mass.
Hogg, Gory	Lewisburg, W. Va.
Holmes, James	Springfield, Mass.
Horky, J. Ralph	Aberdeen, Md.
Hornbrook, K. M.	New Martinsville, W. Va.
Howard, W. L.	Salisbury, Md.
Hubbard, J. Edward	Huntington, W. Va.
Hull, Harry C.	Baltimore, Md.
Hundley, J. Mason, Jr.	Baltimore, Md.
Hundley, John T. T.	Lynchburg, Va.
Hundley, P. G.	Lynchburg, Va.
Hurtt, Harry	Washington, D. C.
Hussey, Raymond	Baltimore, Md.
Hyman, Calvin	Baltimore, Md.

Ingram, David N.	Houston, Pa.
Insley, Philip A.	Salisbury, Md.
Ireland, R. A.	Charleston, W. Va.

Jackson, M. Elliot	New Rochelle, N. Y.
Jackvony, Albert H.	Providence, R. I.
Jacobs, Joye	Baltimore, Md
Jacobs, Morris A.	Baltimore, Md.
Jacobson, Philip	Petersburg, Va.
Jacobson, Samuel	Cumberland, Md.
James, Oliver V.	Milford, Del.
Jarrell, K. M.	Beckley, W. Va.
Jensen, Jacob R.	Sykesville, Md.
Jessurun, Samuel H.	Newark, N. J.
Johns, Benj. C.	Morgantown, W. Va.
Johnson, Henry R.	Fairmont, W. Va.
Johnson, Marius P.	Baltimore, Md.
Johnson, Philip	Fairmont, W. Va.
Jones, C. Henry	Camp Pendleton, Va.
Jones, E. A.	Cambridge, Md.
Judd, C. N.	Philadelphia, Pa.

Kader, Benjamin	Baltimore, Md.
Katz, Albert H.	Charleston, W. Va.
Katz, Simon	Brooklyn, N. Y.
Kaufman, E. W.	Camden, N. J.
Kaufman, Israel	Brooklyn, N. Y.
Kaye, Philip L.	Long Island City, N. Y.
Keesor, Charles H.	Wheeling, W. Va.
Keith, Marion	Greensboro, N. C.
Kelly, M. B.	Wheeling, W. Va.
Kemler, Joseph	Baltimore, Md.
Kerrigan, John J.	Fall River, Mass.
King, W. P.	Weston, W. Va.
Klawans, Maurice	Annapolis, Md.
Klimes, L. F.	Baltimore, Md.
Knox, Joseph C.	Raleigh, N. C.
Kochman, Leon A.	Ellicott City, Md.
Kocyan, Joseph J.	Wilkes-Barre, Pa.
Kohn L. Winfield	New York City
Kohn, Walter	Baltimore, Md.
Krantz, John C., Jr.	Baltimore, Md.
Kraut, Arthur M.	Jersey City, N. J.
Kremen, Abraham	Baltimore, Md.
Kunkel, Anne	Baltimore, Md.
Kurtz, Gerald I.	Paterson, N. J.

Laino, Frank A.	Long Island City, N. Y.
Lake, Lafayette	Cambridge, Mass.
Lancaster, Alston H.	Worcester, Mass.
Langfitt, Frank	Clarksburg, W. Va.
Lanham, A. G.	Ronceverte, W. Va.
Lansdale, P. S.	Frederick, Md.
Lawson, A. F.	Weston, W. Va.
Layman, J. Walter	Hagerstown, Md.
Layton, C. R.	Centreville, Md.
Leibensperger, George F.	Kutztown, Pa.
Lennon, W. E.	Federalsburg, Md.
Lentz, George	York, Pa.
Lenzer, S. P.	Providence, R. I.
Leone, C. B.	Wilmington, Del.
Leskin, Louis W.	Hardeeville, S. C.
Levi, Ernest	Baltimore, Md.
Levy, Charles S.	Baltimore, Md.
Levy, Walter H.	New York City
Lewis, A. C.	Fall River, Mass.
Lichtenberg, Walter	New York City
Lieb, Saul	Newark, N. J.
Lifland, Bernard	Newark, N. J.
Lilly, Goff P.	New York City
Limbach, Earl	New York City
Linger, R. B.	Clarksburg, W. Va.
Linhardt, Elmer	Eastport, Md.
Little, Luther E.	Baltimore, Md.
Lloyd, O. S.	Baltimore, Md.
Longwell, Harold H.	Tyrone, Pa.
Looper, Edward A.	Baltimore, Md.
Louft, R. R.	Charleston, W. Va.
Love, William S., Jr.	Baltimore, Md.
Lubin, Benjamin	Newark, N. J.
Lumpkin, L. U.	Elkins, W. Va.
Lupton, Charles H.	Norfolk, Va.
Lurting, C. W.	Pittsburgh, Pa.
Lusby, Frank	Hagerstown, Md.
Lutz, J. Fletcher	York, Pa.
Lutz, John F.	Baltimore, Md.
Lynch, Raymond A.	Wilmington, Del.

MacGregor, Allan W.......Paterson, N. J.
Mackey, W. K........Huntington, W. Va.
Maconachy, W. V........Baltimore, Md.
Maldeis, Howard J........Baltimore, Md.
Markens, Edward W.......Newark, N. J.
Marsh, W. H............Solomons, Md.
Martin, F. E...New Martinsville, W. Va.
Martin, J. Willis.........Annapolis, Md.
Marton, Samuel......New York City
Matheke, George A........Newark, N. J.
Matheke, Otto G..........Newark, N. J.
Matheke, O. G., Jr.........Newark, N. J.
Mautner, Emil...........Newark, N. J.
Maysels, Alexander J.....Bethlehem, Pa.
Mazer, Maxwell..........Baltimore, Md.
McCain, Paul P.......Sanatorium, N. C.
McCamey, K. E...........Paterson, N. J.
McClung, James......Richwood, W. Va.
McCollum, J. Riley....St. Marys, W. Va.
McCoy, C. Glenn......Wheeling, W. Va.
McDede, Frank F.........Paterson, N. J.
McElwee, Ross........Statesville, N. C.
McGinn, J. F...........Pawtucket, R. I.
McHenry, D. J...............Hellam, Pa.
McLean, George........Baltimore, Md.
McMillan, Wm. A...Charleston, W. Va.
McMillan, W. Owen..Charleston, W. Va.
McPherson, S. D.......Durham, N. C.
Mech, Karl F...........Baltimore, Md.
Medairy, George C...Owings Mills, Md.
Megahan, Burke....Elm Grove, W. Va.
Meister, Aaron..........Hollis, N. Y.
Mendeloff, M. I.....Charleston, W. Va.
Merkel, Walter C.......Baltimore, Md.
Miller, B. H. K.......Philadelphia, Pa.
Miller, Edgar R.......Wilmington, Del.
Miller, Frank O........Ellicott City, Md.
Miller, H. S...........Wilmington, Del.
Miller, James A.........Pikesville, Md.
Mills, L. H...........Clarksburg, W. Va.
Mirow, Richard R.........Merrick, N. Y.
Moore, S. G................Elkins, W. Va.
Morgan, I. J...........Pittsburgh, Pa.
Morgan, Zackariah R.....Baltimore, Md.
Morris, Frank K.......Baltimore, Md.
Morris, S. J.......Morgantown, W. Va.
Morrison, C. F.......Philadelphia, Pa.
Morrison, Samuel......Baltimore, Md.
Mosby, C. V...........St. Louis, Mo.
Moses, Benjamin B.......Baltimore, Md.
Motta, P. G...........Carnegie, Pa.
Moulton, Allen T.........Boston, Mass.
Moyers, M. B.........Mt. Rainier, Md.
Musser, Ruth..........Baltimore, Md.
Muth, John............Baltimore, Md.
Myers, Benjamin F...Chambersburg, Pa.
Myers, Karl............Philippi, W. Va.

Natato, Joseph..........Newark, N. J.
Neller, Walter I.......Middletown, N. Y.
Neustaedter, Theodore....New York City
Newman, Saul........New York City
Nichols, Samuel A.......Clarksville, Md.
Nock, Randolph N.........Salisbury, Md.
Noell, R. H.........Rocky Mount, N. C.
Norris, J. Edward.........Baltimore, Md.
Norris, L. D.......Fairmont, W. Va.
Novak, Emil..........Baltimore, Md.
Novey, M. Alexander....Baltimore, Md.

O'Brien, T. J.......Pawtucket, R. I.
O'Brien, William A.......Passaic, N. J.
O'Connor, James J.....Dickson City, Pa.
Oddo, V. J.............Providence, R. I.
Ogden, Frank N.........Baltimore, Md.
O'Neil, F. P.........Cumberland, Md.
Orem, F. S.............Baltimore, Md.
Orff, J. Henry...........Shillington, Pa.
O'Rourk, Thomas R.....Baltimore, Md.
O'Rourke, Patrick I.....Providence, R. I.
Orr, Wm. J. B.........Washington, D. C.
Oster, Robert H.........Baltimore, Md.
Owens, C. L.........Cumberland, Md.

Pachtman, Isadore......Pittsburgh, Pa.
Palitz, Leo S.............New York City
Panebianco, Richard R.....Corona, N. Y.
Parks, Charles L.......Fairmont, W. Va.
Parr, W. A...........Baltimore, Md.
Pate, F. J...........Greensboro, N. C.
Peake, C. W...........Baltimore, Md.
Pepper, J. K.......Winston-Salem, N. C.
Peters, H. Raymond......Baltimore, Md.
Petrulias, George A.....Bethlehem, Pa.
Phillips, L. D.......Marshallton, Del.
Pincoffs, Maurice C.....Baltimore, Md.
Pink, S. H.................Butler, N. J.
Point, W. W.........Charleston, W. Va.
Pokorny, Joseph..........Baltimore, Md.
Pokrass, Fred P.........Reading, Pa.
Polan, Lake..........Huntington, W. Va.
Polizzotti, Joseph.........Paterson, N. J.
Polsue, Wm. C.........Charleston, W. Va.
Ponte, Joseph P., Jr..New Bedford, Mass.
Porterfield, M. C.......Hampstead, Md.
Porterfield, M. H.....Martinsburg, W. Va.
Post, A. T...........Clarksburg, W. Va.
Post, Cecil O.........Clarksburg, W. Va.
Preston, D. G.........Lewisburg, W. Va.
Preston, J. W.............Roanoke, Va.
Price, J. W.........Marlinton, W. Va.
Price, Samuel J.......Queenstown, Md.
Purvis, J. Oliver......Annapolis, Md.

Quinn, Elwood T.......Jenkintown, Pa.
Quinn, John F..........Bridgeport, Conn.

Rabinowitz, J. Herbert....Newark, N. J.
Racusin, Nathan.........Baltimore, Md.
Radman, Melvin H.......Baltimore, Md.
Rankin, Wm. S.........Charlotte, N. C.
Rascoff, Henry......Brooklyn, N. Y.
Rattenni, Arthur......Providence, R. I.
Ray, John B...........Leaksville, N. C.
Reindollar, William F.....Baltimore, Md.
Resnick, Elton........Wilmington, Del.

Reynolds, Francis	Athol, Mass.
Rhone, David S.	Camden, N. J.
Rich, Benjamin S.	Baltimore, Md.
Richmond, L. C.	Huntington, W. Va.
Ridgely, Irwin O.	Baltimore, Md.
Ries, A. F.	Baltimore, Md.
Riley, John L.	Snow Hill, Md.
Riley, W. M.	Downingtown, Pa.
Roberts, B. W.	Durham, N. C.
Robertson, Edwin M.	Durham, N. C.
Robinson, Harry M., Jr.	Baltimore, Md.
Robinson, Milton	Brooklyn, N. Y.
Roemer, Jacob	Paterson, N. J.
Rohm, J. Z.	Carnegie, Pa.
Rohrer, Caleb W. G.	Baltimore, Md.
Romine, C. C.	Morgantown, W. Va.
Rose, Edward E.	Huntington, W. Va.
Roseman, Ralph B.	Philadelphia, Pa.
Rosen, Morris	Philadelphia, Pa.
Rosenthal, Morris	New York City
Rosenthal, Stephen	Scranton, Pa.
Rousseau, J. P.	Winston-Salem, N. C.
Rowe, Walter B.	Baltimore, Md.
Rowland, J. M. H.	Baltimore, Md.
Rozum, John K.	Cumberland, Md.
Rubinstein, H. S.	Baltimore, Md.
Ruche, H. C.	Philadelphia, Pa.
Russell, Thomas J.	Philadelphia, Pa.
Rynkiewicz, S. H.	Kingston, Pa.
Sardo, Robert	Highland, Md.
Sarnoff, Jack	Long Island City, N. Y.
Sartorius, N. E., Jr.	Pocomoke City, Md.
Sartorius, N. E., Sr.	Pocomoke City, Md.
Sashin, David	New York City
Saunders, Thomas S.	Portland, Ore.
Savage, J. E.	Baltimore, Md.
Sayre, C. T.	Charleston, W. Va.
Scanlan, Thomas F.	Providence, R. I.
Schaeffer, Harry B.	Shillington, Pa.
Schenthal, Joseph	Baltimore, Md.
Schiff, Joseph	Springfield, Mass.
Schmidt, Emil G.	Baltimore, Md.
Schneiman, M. H.	Philadelphia, Pa.
Schoolman, E. R.	Frederick, Md.
Schultz, Louis A.	New York City
Schwartz, Ralph A.	Newark, N. J.
Seaks, George H.	Harrisburg, Pa.
Seegar, J. King B. E.	Baltimore, Md.
Seitz, Clyde	York, Pa.
Selby, George D.	Dawsonville, Md.
Sewell, Stephen	Springlake, N. J.
Seymour, William S.	Easton, Md.
Shamer, Maurice	Baltimore, Md.
Shannon, George E.	Baltimore, Md.
Shaw, Christopher C.	Bellows Falls, Vt.
Shea, John	Bridgeport, Conn.
Shelley, Harry	Baltimore, Md.
Shenker, Morris	Baltimore, Md.
Sherman, Elizabeth	Front Royal, Va.
Shill, Benjamin	Newark, N. J.
Shimanek, Lawrence J.	Baltimore, Md.
Shipley, Arthur M.	Baltimore, Md.
Shochat, A. J.	Baltimore, Md.
Shoemaker, A. B.	Boston, Mass.
Shriver, Joseph K.	Cambridge, Md.
Siegel, Isadore	Baltimore, Md.
Siegel, Milton	New York City
Silver, A. A.	Baltimore, Md.
Simmons, J. Frederick	Cambridge, Md.
Simon, Joseph R.	Pittsburgh, Pa.
Simpers, H. G.	Chestertown, Md.
Singewald, Albert G.	Baltimore, Md.
Skillman, Wilbur F.	Baltimore, Md.
Slusher, Hamilton J.	Frederick, Md.
Smith, Dietrich C.	Baltimore, Md.
Smith, Edward P.	Baltimore, Md.
Smith, Sol	Baltimore, Md.
Smoot, A. C.	Georgetown, Del.
Smoot, M. C.	Hagerstown, Md.
Snaith, Theresa	Weston, W. Va.
Snavely, Earl H.	Newark, N. J.
Snyder, John N.	Masontown, Pa.
Snyder, Nathan	Baltimore, Md.
Snyder, Samuel	Baltimore, Md.
Sollod, Aaron	Baltimore, Md.
Solomon, Simon	Baltimore, Md.
Soltroff, Jack G.	Philadelphia, Pa.
Spaeder, Philip	York, Pa.
Spano, Frank	Union City, N. J.
Sparta, Anthony J.	Easton, Pa.
Speake, Thomas	Baltimore, Md.
Speas, D. C.	Winston-Salem, N. C.
Spelsburg, W. W.	Clarksburg, W. Va.
Spencer, Hugh R.	Baltimore, Md.
Spielman, Morton M.	New York City
Spoon, S. C., Jr.	Burlington, N. C.
Sprague, Edward H.	Newark, N. J.
Sprecher, Milford H.	Elkton, Md.
Squires, Millard F., Jr.	Richardson Park, Del.
Stacy, Theodore E.	Baltimore, Md.
Stansfield, C. W.	Fall River, Mass.
Steele, Guy	Cambridge, Md.
Steenbergen, J. H.	Huntington, W. Va.
Stein, B. M.	Hempstead, N. Y.
Stein, Harold M.	Paterson, N. J.
Steinberg, Samuel	Philadelphia, Pa.
Stern, Morris H.	Clifton, N. J.
Stevens, Russell	Wilkes-Barre, Pa.
Stewart, Charles W.	Baltimore, Md.
Stewart, F. C.	Manchester, N. H.
Stonestreet, W. W.	Morgantown, W. Va.
Stovin, Joseph S.	New York City
Sturgis, R. W.	Norfolk, Va.
Sutton, H. Lawrence	Newark, N. J.
Swartzwelder, W. R.	York, Pa.
Swint, B. H.	Charleston, W. Va.
Swiss, A. G.	Parkersburg, W. Va.
Talbott, James E.	Baltimore, Md.
Taylor, Francis N.	Petersburg, Va.
Taylor, J. T.	Greensboro, N. C.
Taylor, Robert B.	Baltimore, Md.
Teagarden, E. V.	Baltimore, Md.
Teitelbaum, H. A.	Baltimore, Md.

Thomas, B. O.	Frederick, Md.
Thomas, Edward P.	Frederick, Md.
Thompson, C. T.	Morgantown, W. Va.
Thompson, James U.	Baltimore, Md.
Tollin, Louis	Baltimore, Md.
Tolson, Howard L.	Cumberland, Md.
Toulson, W. Houston	Baltimore, Md.
Trevaskis, R. W.	Cumberland, Md.
Truitt, C. R.	Salisbury, Md.
Trynin, Aaron H.	Brooklyn, N. Y.
Tuckwiller, J. R.	Fairmont, W. Va.
Tull, Myron	Baltimore, Md.

Uhlenhuth, Eduard	Baltimore, Md.
Ullrich, Henry F.	Baltimore, Md.
Ullrich, Henry J.	Baltimore, Md.

Valentine, A. W.	Washington, D. C.
Van Metre, J. L.	Charles Town, W. Va.
Van Ormer, W. Alfred	Cumberland, Md.
Varney, W. H.	Washington, N. J.
Vieweg, G. L., Jr.	Wheeling, W. Va.
Vogel, S. Zachary	Woodhaven, N. Y.
Volenick, Lee	Baltimore, Md.
Von Schultz, Augustus	Baltimore, Md.

Wack, Frederick	Baltimore, Md.
Walker, R. H.	Charleston, W. Va.
Wallack, Charles A.	Newark, N. J.
Walton, Henry J.	Baltimore, Md.
Wanner, Jesse R., Sr.	Salisbury, Md.
Warman, W. M.	Morgantown, W. Va.
Warner, C. Gardner	Baltimore, Md.
Warren, B. P.	Laurel, Md.
Warren, John M.	Laurel, Md.
Wassersweig, M. W.	Reading, Pa.
Waters, Zach J.	Martinsburg, W. Va.
Waterworth, S. J.	Clearfield, Pa.
Weber, John J.	Pittsburgh, Pa.
Weinert, Henry	Passaic, N. J.
Weintraub, Fred S.	Pittsburgh, Pa.
Welch, Robert S. G.	Annapolis, Md.
Wells, Gibson J.	Baltimore, Md.
Weltner, F. P.	Charleston, W. Va.
Welton, William A.	Fairmont, W. Va.
Wentz, P. N.	York, Pa.
Werner, A. S.	Brooklyn, N. Y.
Wheeler, H. Lawrence	Baltimore, Md.
Wheltle, Charles B.	Baltimore, Md.
Whisler, H. Allen	Clarksburg, W. Va.
White, E. W.	Poolesville, Md.
Widmeyer, Robt. S.	Parkersburg, W. Va.
Wilkinson, Albert L.	Baltimore, Md.
Williams, J. F., Jr.	Clarksburg, W. Va.
Williams, J. F., Sr.	Clarksburg, W. Va.
Williams, L. V.	York, Pa.
Williams, Mortimer	Roanoke, Va.
Williams, Wm. F.	Cumberland, Md.
Wilner, Joseph W.	New York City
Wilson, Frank M.	Cumberland, Md.
Wilson, J. E.	Elkins, W. Va.
Wilson, Mark S.	Marlinton, W. Va.
Wilson, S. C.	Oxford, Pa.
Winters, Walter M.	Paterson, N. J.
Wise, Francis R.	York, Pa.
Wise, Walter D.	Baltimore, Md.
Wolff, Eldridge H.	Cambridge, Md.
Wolff, Thomas C.	Baltimore, Md.
Wollenweber, H. L.	Baltimore, Md.
Wood, Everet H.	Auburn, N. Y.
Woodall, R. E.	Charleston, W. Va.
Woolley, Lawrence F.	Baltimore, Md.
Wright, Eugene B.	Clarksburg, W. Va.
Wriston, Robert	Beckley, W. Va.
Wurtzel, Milton	Newark, N. J.
Wylie, H. Boyd	Baltimore, Md.

Yaeger, Leslie A.	Trenton, N. J.
Yeager, W. Howard	Hagerstown, Md.
Young, Ralph F.	Williamsport, Md.
Yubas, Morris L.	Philadelphia, Pa.

Zager, Saul	Newark, N. J.
Zaslow, John	Brooklyn, N. Y.
Zimmerman, F. T.	Canandaigua, N. Y.
Zinberg, I. S.	Baltimore, Md.
Zinn, Waitman F.	Baltimore, Md.
Zurawski, Charles	Providence, R. I.

Julius Friedenwald

JULIUS FRIEDENWALD, M.A., M.D., 1866–1941

For eighty years the name of Friedenwald in Baltimore has been closely associated with the best in medicine. The founder of this medical clan was Aaron, a University of Maryland graduate and an understudy of Dr. Nathan R. Smith, who later won distinction in the field of ophthalmology, being head of that department in the College of Physicians and Surgeons of which he was a founder.

The eldest son of Aaron was Harry, who followed his father's specialty, in which he has achieved fame and in which he is still active. The second son, the subject of this memoir, was Julius, who became a leader in the field of gastro-enterology. A third brother to become a doctor was Edgar, who has specialized with distinction in pediatrics and who is Professor of Clinical Pediatrics at the University of Maryland and Chief Pediatrician at Mercy Hospital.

In the third generation, there is Jonas, son of Harry, who, like his father and grandfather, is an outstanding ophthalmologist.

Dr. Julius Friedenwald was born in Baltimore on December 20, 1866. He was educated at a private school, the Baltimore City College and later, in 1887, received the degree of Bachelor of Arts from the Johns Hopkins University. He was graduated by the College of Physicians and Surgeons in 1890 and served a two year house officership in what is now Mercy Hospital. Later he studied in Berlin, Paris, Vienna and London and formed friendships and connections that lasted throughout his life. Dr. Friedenwald very promptly developed a large practice, which soon became of unusual proportions. His administration to many patients, however, did not prevent a constant flow of medical papers, monographs, chapters in textbooks and encyclopedias. He wrote two treatises with Dr. John Ruhrah on diet, several smaller books on various subjects in which he was interested, the last being in collaboration with Samuel Morrison the younger of the two brothers associated with him. These were on "Functional Gastro-Enterological Conditions." He was author and co-author of more than 200 articles. His prolific pen kept active until his final illness.

An indication of his early recognition was an honorary degree of Master of Arts conferred by Loyola College in 1898. At the College of Physicians and Surgeons, the School of Medicine of the University of Maryland, and at Mercy Hospital his rise was rapid. By 1909 he was Professor of Gastro-Enterology, a position held until 1935. From 1921 until 1935 he was a member of the Council of the Medical School. He was Gastro-Enterologist-in-Chief of Mercy Hospital and was on the visiting staffs of the University, Sinai and Union Memorial Hospitals, the Church Home and Infirmary, and the Hospital for the Women of Maryland. Dr. Friedenwald was a trustee of the Institute of Advanced Study at Princeton University and a trustee of the Palestine Economic Foundation. He was a member and past President of the Baltimore City Medical Society, a member and a past President of the American Gastro-Enterological Association, and a member of the American College of Physicians, the Association of American Physicians, the Therapeutic Society, the Southern Medical Association, and the Medical and Chirurgical Faculty of Maryland. He was a medalist of the Phi Lambda Kappa fraternity and a member of the Phi Beta Kappa. He belonged to the Johns Hopkins, University, Research, and Phoenix Clubs.

Last year Dr. Friedenwald founded and endowed a Julius Friedenwald medal to be awarded annually by the American Gastro-Enterological Association for distinguished service in that branch of medicine. The first award was made this year to Dr. Walter B. Cannon, Professor of Physiology at Harvard University. It was a great distress to the donor that, because of illness, he could not personally present the medal at the Atlantic City Meeting of the American Gastro-Enterological Association in May.

In 1900 Dr. Friedenwald married Miss Esther Lee Rohr and for forty years they were familiar figures at the opera, the theatre, social functions, and civic activities. Their home on Charles Street was a center of much charming entertaining.

It must have been a great satisfaction to Dr. Friedenwald to have been able to do, day after day, the amount of work he did; to carry to so many people, in all walks of life, so much kindness, affability, optimism and imperturbability; all were mixed with great charity where this was needed. The equanimity extolled by Osler was a fundamental part of his character and it took great wrong or injustice to disturb his calm. Two things disturbed him more than most—racial prejudice, and neglect or poor care of his patients. In these instances his remonstrations were mild but left no doubt as to their sincerity and firmness.

Dr. Friedenwald was a man rather set apart, somewhat aloof from the common herd. While sympathetic, kind and charitable to a high degree, he had few or, one might say, none of the usual faults and weaknesses. This is testified to by his boyhood companions, his medical school classmates and close associates throughout life. It is said by intimate colleagues that they never heard him raise his voice in anger nor heard him say a mean or disagreeable thing about any person. No man made greater effort to do right with more success.

The medical achievements of a physician of Dr. Friedenwald's prominence will be evaluated by others. To those who knew him well he seemed an outstanding example of an energetic, unusually successful man, able to meet the problems and worries of an extremely large practice without becoming in the least hardened, keeping his underlying gentleness of disposition and character through a long life and a painful illness to the last.

"David describeth a citizen of Zion" as "He that walketh uprightly, and worketh righteousness, and speaketh the truth in his heart.

"He that backbiteth not with his tongue, nor doeth evil to his neighbor, nor taketh up a reproach against his neighbor."—Psalm 15, v. 2–3.

Such a man was Julius Friedenwald.

WALTER D. WISE, M.D.

W. C. MITCHELL, M.D.

Dr. W. C. Mitchell, U. of Md. 1889, aged 72, died in Denver, Colorado on March 31, 1941.

Dr. Mitchell was born in Baltimore on November 1, 1869 and attended West Nottingham Academy before entering the University of Maryland. After receiving his degree in medicine he took postgraduate courses at the University of Berlin and the Pasteur Institute. In 1897 he became active in public health work in Denver, where he was appointed City bacteriologist. At his death he was director of laboratories of the State Division of Public Health.

RAMSON R. NORRIS, M.D.

Dr. Ramson R. Norris, U. of Md. 1904, aged 60, died in Crisfield, Maryland on June 9, 1941 of a heart attack.

Dr. Norris was born in Washington, D. C. on October 18, 1881. He was educated at Emerson Institute, Washington, and at George Washington University. After being graduated from the School of Medicine of the University of Maryland and taking a postgraduate course at the Johns Hopkins University, he was appointed to the staff of the old Bay View Hospital in Baltimore. Later he resigned to accept an appointment as assistant surgeon to the old United Railway Company and also served as surgeon to the Baltimore Police Department.

In 1905 Dr. Norris moved to Crisfield and took up the practice of general medicine and surgery there.

During the World War he commanded Field Hospital No. 36 in the Puvennel sector with the Second Army in France. After the war he was acting assistant surgeon in the United States Public Health Service and examiner for the United States Veterans' Bureau. He was a member of the surgical staff of Edward W. McCready Memorial Hospital, and of the Somerset County Welfare Board.

Babb, Benjamin Franklin, Ivor, Va.; class of 1892; aged 76; died, March 31, 1941, of heart disease.

Boose, Theodore, Williamsport, Md.; P. & S., class of 1889; aged 76; died, January 11, 1941, of coronary occlusion and arteriosclerosis.

Collins, Rollins P., Bishopville, Md.; class of 1890; aged 76; died, January 20, 1941, of cerebral hemorrhage.

Cook, Frank M., Hackettstown, N. J.; P. & S., class of 1883; aged 82; died, January 18, 1941, of arteriosclerosis.

Dare, Frank Theodore, Wellsburg, W. Va.; B.M.C., class of 1901; aged 64; died, January 21, 1941, of carcinoma.

Delcher, Hazlett Austin, Toledo, Ohio; class of 1907; aged 56; died, March 3, 1941, of pneumonia.

Dorsey, John Horatio, Grantsville, Md.; class of 1885; veteran of the Spanish-American War; aged 89; died, April 10, 1941, of chronic myocarditis.

Duvally, Frank A., Fall River, Mass.; P. & S., class of 1910; aged 56; died, February 4, 1941, of carcinoma of the esophagus.

Edmondson, Henry Turner, Moultrie, Ga.; P. & S., class of 1911; served during the World War; aged 53; died, January 19, 1941, of myocarditis.

Fleetwood, Edgar Allen, Clarksburg, W. Va.; class of 1904; aged 59; died, February 13, 1941; of injuries received in an automobile accident.

Jackson, William Andrew, Monteagle, Tenn.; P. & S., class of 1896; aged 77; died, March 9, 1941, of myocarditis and aortic regurgitation.

Judkins, Eugene H., Melrose, Mass.; B.M.C., class of 1892; aged 78; died, February 1, 1941, of arteriosclerotic heart disease.

Kemerer, Merion E., Danielsville, Pa.; P. & S., class of 1897; aged 66; died, February 11, 1941, of hypertrophic cirrhosis of the liver.

King, George Robert Seeber, Lake Butler, Fla.; P. & S., class of 1901; aged 63; died, February 28, 1941.

Little, Adolphus Lamar, New Orleans, La.; class of 1910; served during the World War; aged 56; died, January 7, 1941, of coronary occlusion.

McCallum, Malcolm James, Fairfield, Conn.; B.M.C., class of 1901; aged 62; died, January 3, 1941, of pulmonary embolism and mitral stenosis.

McDede, Joseph Searle, Jersey City, N. J.; P. & S., class of 1905; aged 67; died, January 28, 1941, of uremia.

McLenahan, Thomas Morrow, Pittsburgh, Pa.; P. & S., class of 1893; aged 74; died, February 28, 1941, of coronary thrombosis.

MacSherry, Henry Clinton, Baltimore, Md.; class of 1872; aged 89; died, January 9, 1941, of inguinal hernia.

Miller, Abram M., Hyndman, Pa.; B.M.C., class of 1905; aged 71; died, March 4, 1941, of coronary thrombosis.

Miller, Elmore R., Ephrata, Pa.; class of 1892; aged 72; died, March 4, 1941, of coronary occlusion.

Mitchell, William C., Denver, Colo.; class of 1889; aged 72; died, March 31, 1941, of coronary thrombosis.

Moor, Frederick Clifton, Tallahassee, Fla.; class of 1903; served during the World War; aged 61; died, February 18, 1941.

Nalley, Harry, Mt. Rainier, Md.; class of 1900; aged 64; died, February 7, 1941.

Perry, Thomas B., Miami, Fla.; P. & S., class of 1885; veteran of the Spanish-American War; aged 77; died, March 8, 1941, of acute dilatation of the heart.

Pierce, Franklin, Norfolk, Va.; P. & S., class of 1875; aged 87; died, January 18, 1941, of bronchopneumonia and arteriosclerosis.

Salton, Russell Arthur, Williamson, W. Va.; B.M.C., class of 1911; served during the World War; aged 53; died, January 20, 1941, of pulmonary embolism.

Shands, Aurelius R., Washington, D. C.; class of 1884; aged 80; died, April 29, 1941, of a heart attack.

Stathers, Walter E., Buckhannon, W. Va.; P. & S., class of 1881; Civil War veteran; aged 92; died, January 19, 1941.

Townsend, William Guy, Baltimore, Md.; class of 1888; aged 76; died, March 12, 1941, of carcinoma of the prostate.

Vogler, George C. E., Frederick, Md.; B.M.C., class of 1894; aged 76; died, February 8, 1941, of coronary thrombosis.

Walker, James Taylor, Richmond, Va.; P. & S., class of 1886; aged 81; died, March 12, 1941.

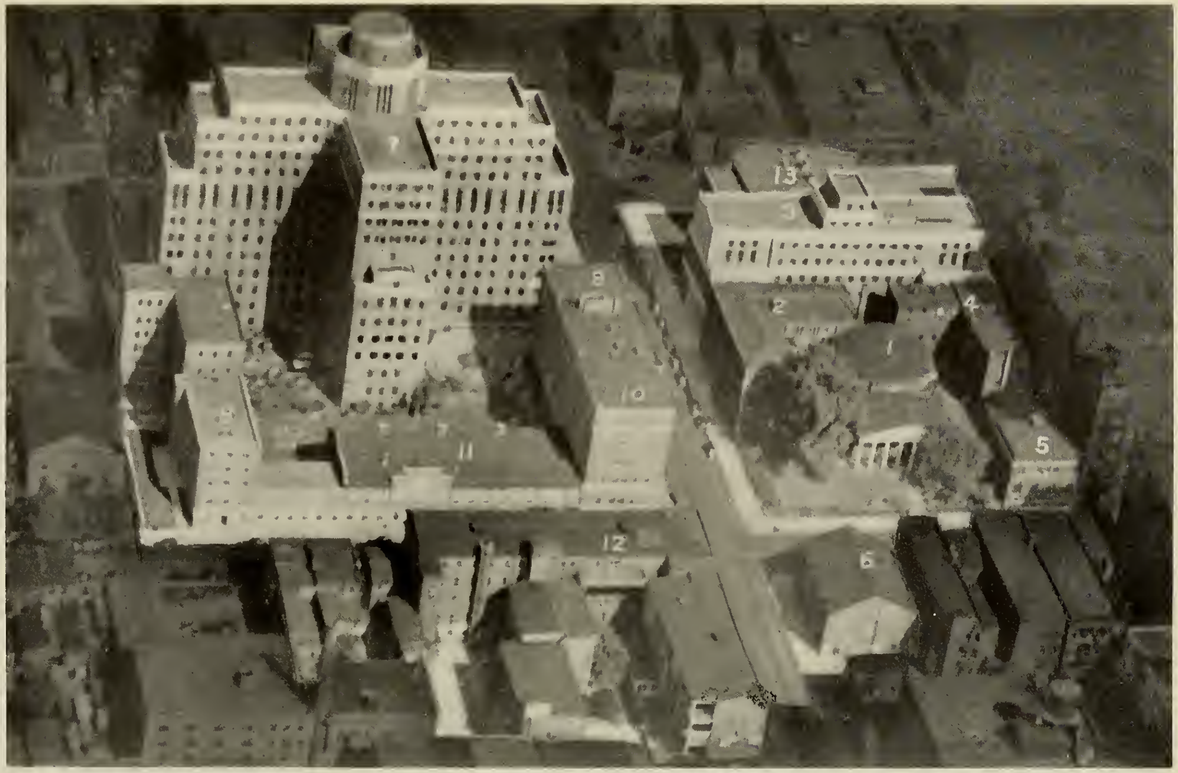
Watkins, William C., Oconto, Wis.; B.M.C., class of 1905; served during the World War; aged 61; died, March 19, 1941.

White, Joseph Augustus, Richmond, Va.; class of 1869; aged 92; died, February 16, 1941, of myocarditis.

Wimberley, George Lewis, Rocky Mount, N. C.; class of 1883; died, March, 1941.

Windsor, William S., Climax Springs, Mo.; class of 1890; aged 79; died, March 14, 1941.

A Section of the Students' Laboratory in the Department of Pharmacology



Baltimore Schools—University of Maryland

1. Original Medical Building
2. Laboratory Building, Medicine
3. Bressler Building, Medicine
4. Gray Laboratory, Students' Lounge, Medicine
5. Administration Building, College of Education, Baltimore Division
6. Medical Library
7. University Hospital
8. Nurses' Home, Medicine
9. School of Pharmacy
10. School of Dentistry
11. Dental Clinic
12. Out-Patient Clinics, Medicine
13. School of Law

BULLETIN
OF THE
SCHOOL *of* MEDICINE
UNIVERSITY OF MARYLAND

1807-1856-1920

Catalogue and Announcement

The One Hundred Thirty-Fifth Annual Session

1941-1942

VOLUME 26, NO. 2
SEPTEMBER, 1941

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1941

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JANUARY

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JUNE

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JULY

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AUGUST

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SEPTEMBER

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OCTOBER

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NOVEMBER

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DECEMBER

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1943

JANUARY

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FEBRUARY

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MARCH

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APRIL

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JUNE

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CALENDAR 1941–42, SCHOOL OF MEDICINE

1941		
September 18–20		Conditioned Examinations

FIRST SEMESTER

September 23	Tuesday	*Registration, freshmen and sophomores
September 24	Wednesday	*Registration, all other students
September 25	Thursday	Instruction begins
November 19	Wednesday	Instruction suspended at 5:00 P.M. Thanksgiving Holidays
November 24	Monday	Instruction resumed
December 20	Saturday	Instruction suspended at 2:00 P.M. Christmas Holidays
1942		
January 5	Monday	Instruction resumed
January 24	Saturday	Instruction suspended at 2:00 P.M.
January 26 to January 31	Monday Saturday	Examinations, all classes, and *Registration for second semester
January 31	Saturday	First semester completed at 12:30 P.M.

SECOND SEMESTER

February 2	Monday	Instruction resumed
February 21	Saturday	Instruction suspended at 2:00 P.M.
February 23	Monday	Holiday—Washington's Birthday
February 24	Tuesday	Instruction resumed
April 1	Wednesday	Instruction suspended at 5:00 P.M. Easter Holidays
April 8	Wednesday	Instruction resumed
May 16	Saturday	Senior and junior instruction ends at 2:00 P.M.
May 18	Monday	Senior and junior examinations begin
May 23	Saturday	Senior examinations completed Sophomore and freshman instruction ends at 12:00 M.
May 25	Monday	Junior examinations continue Sophomore and freshman examinations begin
May 30	Saturday	Announcement of graduates All examinations completed Second semester completed at 12:30 P.M.
June 6	Saturday	Commencement at 11:00 A.M.

PARTIAL CALENDAR FOR 1942–1943

1942		
September 17–19		Conditioned Examinations
September 22	Tuesday	*Registration, freshmen and sophomores
September 23	Wednesday	*Registration, all other students
September 24	Thursday	Instruction begins

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* A student who fails to register prior to or within the day or days specified will be called upon to pay a late registration fee of five dollars ($5.00). The last day of registration with fee added to regular charges is Saturday of the week in which registration begins.

The offices of the registrar and comptroller are open daily, not including Saturday, from 9:00 a.m. to 5:00 p.m., and Saturday from 9:00 a.m. to 12:30 p.m.

Early registration is encouraged.

ORGANIZATION

THE UNIVERSITY OF MARYLAND

Harry Clifton Byrd, B.S., LL.D., D.Sc., *President and Executive Officer*

BOARD OF REGENTS

	Term expires
Henry Holzapfel, Jr., *Chairman* Hagerstown, Washington County	1943
Mrs. John L. Whitehurst, *Secretary* 4101 Greenway, Baltimore	1947
J. Milton Patterson, *Treasurer* 1015 Argonne Drive, Baltimore	1944
William P. Cole, Jr. Towson, Baltimore County	1949
W. Calvin Chesnut Roland Park, Baltimore	1942
John E. Semmes 100 W. University Parkway, Baltimore	1942
Rowland K. Adams 1808 Fairbank Road, Baltimore	1948
Philip C. Turner Parkton, Baltimore county	1950

The control of the University of Maryland is assigned to a Board of Regents, appointed by the Governor and confirmed by the Senate for terms of nine years each. The general administration of the University is vested in the President. The University Council is an advisory body, composed of the President, the Director of the Agricultural Experiment Station, the Director of the Extension Service, and the Deans. The University Council acts upon all matters having relation to the University as a whole, or to co-operative work between the constituent groups. Each school has its own Faculty Council, composed of the Dean and members of its faculty; each Faculty Council controls the internal affairs of the group it represents.

The University has the following educational organizations:

At Baltimore

The School of Dentistry
The School of Law
The School of Medicine
The School of Nursing
The School of Pharmacy

At College Park

The College of Agriculture
The College of Arts and Sciences
The College of Commerce
The College of Education
The College of Engineering
The College of Home Economics
The Graduate School
The Department of Military Science and Tactics
The Department of Physical Education and Recreation
The Summer School

THE UNIVERSITY SENATE
(Baltimore Schools)

H. C. Byrd, B.S., LL.D., D.Sc. . . . . President of the University
H. Boyd Wylie, M.D. . . . . Acting Dean of the School of Medicine
Roger Howell, Ph.D., LL.B . . . . Dean of the School of Law
Andrew G. DuMez, M.S., Ph.D . . . . Dean of the School of Pharmacy
J. Ben Robinson, D.D.S., F.A.C.D . . . . Dean of the School of Dentistry
L. B. Broughton, Ph.D . . . . Dean of the College of Arts and Sciences
John E. Savage, B.S., M.D . . . . Acting Superintendent of the Hospital
Alma H. Preinkert, M.A . . . . Registrar
Edgar F. Long, Ph.D . . . . Acting Director of Admissions

FACULTY OF PHYSIC

EMERTI

J. Frank Crouch, M.D. . . . Professor of Clinical Ophthalmology and Otology, Emeritus
Harry Friedenwald, A.B., M.D., D.H.L., D.Sc. . Professor of Ophthalmology, Emeritus
William S. Gardner, M.D. . . . . Professor of Gynecology, Emeritus
§Julius Friedenwald, M.A., M.D . . . . Professor of Gastro-Enterology, Emeritus
J. M. H. Rowland, M.D., D.Sc., LL.D.
Professor of Obstetrics, Emeritus; Dean, Emeritus
Henry J. Walton, M.D. . . . . Professor of Roentgenology, Emeritus

MEDICAL COUNCIL

H. Boyd Wylie, *Acting Dean*

Arthur M. Shipley
Hugh R. Spencer
Carl L. Davis
Maurice C. Pincoffs
Frank W. Hachtel
Eduard Uhlenhuth

Clyde A. Clapp
John C. Krantz, Jr.
Walter D. Wise
J. Mason Hundley, Jr.
William R. Amberson
Louis H. Douglass

PROFESSORS

Arthur M. Shipley, M.D., Sc.D., Professor of Surgery.
Hugh R. Spencer, M.D., Professor of Pathology.
H. Boyd Wylie, M.D., Professor of Biological Chemistry and Acting Dean.
Carl L. Davis, M.D., Professor of Anatomy.
Maurice C. Pincoffs, B.S., M.D., Professor of Medicine.
Frank W. Hachtel, M.D., Professor of Bacteriology.
Clyde A. Clapp, M.D., Professor of Ophthalmology.
Eduard Uhlenhuth, Ph.D., Professor of Anatomy.

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§ Died, June 8, 1941.

On the lists of the Faculty of Physic and Fellows, and of the Hospital and Dispensary staffs, are given the names and positions assigned during the sessions 1940–1941 and 1941–1942, unless otherwise indicated. Interim changes are noted as follows:
★ On leave, military service.
* Promotions effective 1941–1942. † Appointments effective 1941–1942.
‡ Resignations effective during or at the end of 1940–1941. § Deaths—Dates of.

John C. Krantz, Jr., Ph.D., Professor of Pharmacology.
J. Mason Hundley, Jr., M.A., M.D., Professor of Gynecology.
Walter D. Wise, M.D., Professor of Surgery.
William R. Amberson, Ph.D., Professor of Physiology.
Louis H. Douglass, M.D., Professor of Obstetrics.
Joseph E. Gichner, M.D., Professor of Clinical Medicine and Physical Therapeutics.
Irving J. Spear, M.D., Professor of Neurology.
Charles F. Blake, M.A., M.D., Professor of Diseases of the Rectum and Colon.
S. Griffith Davis, M.S., M.D., Professor of Anaesthesia.
G. Carroll Lockard, M.D., Professor of Clinical Medicine.
Harvey G. Beck, M.D., D.Sc., Professor of Clinical Medicine.
Albertus Cotton, M.A., M.D., Professor of Orthopaedic Surgery and Roentgenology.
Andrew C. Gillis, M.A., M.D., LL.D., Professor of Neurology.
‡Henry J. Walton, M.D., Professor of Roentgenology.
Ross McC. Chapman, M.D., Professor of Psychiatry.
Edgar B. Friedenwald, M.D., Professor of Clinical Pediatrics.
Edward A. Looper, M.D., D.Oph., Professor of Diseases of the Nose and Throat.
J. W. Downey, M.D., Professor of Otology.
§Harry M. Stein, M.D., Professor of Clinical Medicine.
W. H. Toulson, M.Sc., M.D., Professor of Genito-Urinary Surgery.
Charles Bagley, Jr., M.A., M.D., Professor of Neurological Surgery.
Allen Fiske Voshell, A.B., M.D., Professor of Orthopaedic Surgery.
Page Edmunds, M.D., Professor of Traumatic Surgery.
Harry M. Robinson, M.D., Professor of Dermatology.
Elliott H. Hutchins, M.A., M.D., Professor of Clinical Surgery.
Charles Reid Edwards, M.D., Professor of Clinical Surgery.
F. L. Jennings, M.D., Professor of Clinical Surgery.
Huntington Williams, M.D., Dr.P.H., Professor of Hygiene and Public Health.
C. Loring Joslin, M.D., Professor of Pediatrics.
Brice M. Dorsey, D.D.S., Professor of Oral Surgery.
J. Dawson Reeder, M.D., Professor of Diseases of the Rectum and Colon.
Thomas P. Sprunt, A.B., M.D., Professor of Clinical Medicine.
*Thomas B. Aycock, B.S., M.D., Professor of Clinical Surgery.
*Walter L. Kilby, M.D., Acting Professor of Roentgenology.
Compton Riely, M.D., Clinical Professor of Orthopaedic Surgery.
§W. S. Smith, M.D., Clinical Professor of Gynecology.
M. Randolph Kahn, M.D., Clinical Professor of Ophthalmology.
Waitman F. Zinn, M.D., Clinical Professor of Diseases of the Nose and Throat.
A. J. Gillis, M.D., Clinical Professor of Genito-Urinary Surgery.
Theodore H. Morrison, M.D., Clinical Professor of Gastro-Enterology.
Abram S. Samuels, A.B., M.D., Clinical Professor of Gynecology.
H. K. Fleck, M.D., Clinical Professor of Ophthalmology.
*‡Monte Edwards, M.D., *Clinical Professor of Diseases of the Rectum and Colon and ‡Associate Professor of Surgery.
*D. J. Pessagno, A.B., M.D., Clinical Professory of Surgery.
*Kenneth D. Legge, M.D., Clinical Professor of Genito-Urinary Surgery.

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§ Dr. Stein: Died November 15, 1940.
§ Dr. Smith: Died December 9, 1940.

ASSOCIATE PROFESSORS

R. W. Locher, M.D., Associate Professor of Clinical Surgery.
H. R. Peters, A.B., M.D., Associate Professor of Medicine.
T. Nelson Carey, M.D., Associate Professor of Medicine and Physician in Charge of Medical Care of Students.
Howard J. Maldeis, M.D., Associate Professor of Medical Jurisprudence and Associate in Pathology.
G. M. Settle, A.B., M.D., Associate Professor of Neurology and Clinical Medicine.
Thomas R. Chambers, A.B., M.D., Associate Professor of Surgery.
William H. Smith, M.D., Associate Professor of Clinical Medicine.
Paul W. Clough, B.S., M.D., Associate Professor of Medicine.
Sydney R. Miller, B.S., M.D., Associate Professor of Medicine.
J. McFarland Bergland, B.S., M.D., Associate Professor of Obstetrics.
Walter A. Baetjer, A.B., M.D.. Associate Professor of Medicine.
Edward S. Johnson, M.D., Associate Professor of Surgery.
Emil Novak, A.B., M.D., D.Sc., Associate Professor of Obstetrics.
Ralph P. Truitt, M.D., Associate Professor of Psychiatry.
Moses Gellman, B.S., M.D., Associate Professor of Orthopaedic Surgery.
Harry L. Rogers, M.D., Associate Professor of Orthopaedic Surgery.
O. G. Harne, Associate Professor of Histology.
Charles A. Reifschneider, M.D., Associate Professor of Traumatic Surgery and Oral Surgery.
Frank H. J. Figge, Ph.D., Associate Professor of Gross Anatomy.
Wm. S. Love, Jr., A.B., M.D., Associate Professor of Medicine and Instructor in Pathology.
Raymond Hussey, M.A., M.D., Associate Professor of Medicine.
Louis A. M. Krause, M.D., Associate Professor of Medicine.
N. Clyde Marvel, A.B., M.D., Associate Professor of Surgery.
T. Campbell Goodwin, M.S., M.D., Associate Professor of Pediatrics.
Carl Dame Clarke, M.A., Associate Professor of Art as Applied to Medicine.
Emil G. Schmidt, Ph.D., LL.B., Associate Professor of Biological Chemistry.
Lawrence F. Woolley, M.D., Associate Professor of Psychiatry.
Richard G. Coblentz, M.A., M.D., Associate Professor of Neurological Surgery.
†Grant E. Ward, A.B., M.D., Associate Professor of Surgery and †Oral Surgery.
Dietrich Conrad Smith, Ph.D., Associate Professor of Physiology.
Franklin B. Anderson, M.D., Associate Professor of Diseases of the Nose and Throat and Otology.
Leon Freedom, M.D., Associate Professor of Neurology and Instructor in Pathology.
James G. McAlpine, Ph.D., Associate Professor of Bacteriology.
C. Jelleff Carr, Ph.D., Associate Professor of Pharmacology.
Cyrus F. Horine, M.D., Associate Professor of Surgery.
*Robert B. Wright, B.S., M.D., Associate Professor of Pathology.
*C. Gardner Warner, A.B., M.D., Associate Professor of Pathology.

ASSISTANT PROFESSORS

George McLean, M.D., Assistant Professor of Medicine.
Maurice Feldman, M.D., Assistant Professor of Gastro-Enterology.
John H. Traband, M.D., Assistant Professor of Pediatrics.
Albert Jaffe, M.D., Assistant Professor of Pediatrics.
★Harry Goldsmith, M.D., Assistant Professor of Psychiatry.

H. W. Newell, M.D., Assistant Professor of Psychiatry.
Zachariah Morgan, M.D., Assistant Professor of Gastro-Enterology.
Samuel Morrison, A.B., M.D., Assistant Professor of Gastro-Enterology.
Harry M. Murdock, B.S., M.D., Assistant Professor of Psychiatry.
A. H. Finkelstein, M.D., Assistant Professor of Pediatrics.
John E. Legge, M.D., Assistant Professor of Medicine.
Thomas C. Wolff, Litt.B., M.D., C.M., Assistant Professor of Medicine.
Frederick B. Smith, M.D., Assistant Professor of Pediatrics.
J. G. M. Reese, M.D., Assistant Professor of Obstetrics.
M. Alexander Novey, A.B., M.D., Assistant Professor of Obstetrics.
Thomas K. Galvin, M.D., Assistant Professor of Gynecology.
Leo Brady, A.B., M.D., Assistant Professor of Gynecology.
R. G. Willse, M.D., Assistant Professor of Gynecology.
George A. Strauss, Jr., M.D., Assistant Professor of Gynecology.
Robert H. Oster, Ph.D., Assistant Professor of Physiology.
H. M. Bubert, M.D., Assistant Professor of Medicine.
Walter C. Merkel, A.B., M.D., Assistant Professor of Pathology.
John F. Lutz, A.B., M.D., Assistant Professor of Histology.
Benjamin Pushkin, M.D., Assistant Professor of Neurology.
William Ellsworth Evans, Ph.D., Assistant Professor of Pharmacology.
H. F. Bongardt, M.D., Assistant Professor of Surgery.
J. Edmund Bradley, B.S., M.D., Assistant Professor of Pediatrics.
George H. Yeager, B.S., M.D., Assistant Professor of Surgery.
Isadore A. Siegel, A.B., M.D., Assistant Professor of Obstetrics.
William H. F. Warthen, A.B., M.D., Assistant Professor of Hygiene and Public Health.
Francis Ellis, A.B., M.D., Assistant Professor of Dermatology.
*Edward P. Smith, M.D., Assistant Professor of Gynecology.
*J. S. Eastland, A.B., M.D., Assistant Professor of Medicine.
*Henry F. Ullrich, M.D., D.Sc., Assistant Professor of Orthopaedic Surgery.
*D. James Greiner, B.S., M.D., Assistant Professor of Pathology.
*James C. Plagge, Ph.D., Assistant Professor of Gross Anatomy.
*†Milton S. Sacks, B.S., M.D., *Assistant Professor of Medicine, Instructor in Pathology, and †Assistant in Bacteriology.
*Ross Davies, M.D., M.P.H., Assistant Professor of Hygiene and Public Health.
*J. Herbert Wilkerson, M.D., Assistant Professor of Oral Surgery.

LECTURERS

Jonas Friedenwald, M.A., M.D., Lecturer in Ophthalmic Pathology.
E. B. Freeman, B.S., M.D., Lecturer in Medicine.
Charles R. Goldsborough, M.A., M.D., Lecturer in Medicine.
W. N. Bispham, Col., M.C., U. S. A. (ret.), Lecturer in Medicine
Leslie B. Hohman, M.D., Lecturer in Psychiatry.

ASSOCIATES

John R. Abercrombie, A.B., M.D., Associate in Dermatology.
Joseph I. Kemler, M.D., Associate in Ophthalmology.
Frank N. Ogden, M.D., Associate in Biological Chemistry.
Austin H. Wood, M.D., Associate in Genito-Urinary Surgery.
Albert E. Goldstein, M.D., Associate in Pathology.
Samuel S. Glick, M.D., Associate in Pediatrics.
‡William J. Todd, M.D., Associate in Pediatrics.

Clewell Howell, B.S., M.D., Associate in Pediatrics.
Joseph Sindler, M.D., Associate in Gastro-Enterology.
I. O. Ridgley, M.S., M.D., Associate in Surgery.
R. D. West, M.D., Associate in Ophthalmology.
Eugene L. Flippin, M.D., Associate in Roentgenology.
L. J. Millan, M.D., Associate in Genito-Urinary Surgery.
W. Raymond McKenzie, M.D., Associate in Diseases of the Nose and Throat.
‡E. P. H. Harrison, A.B., M.D., Associate in Obstetrics.
L. K. Fargo, M.D., Associate in Genito-Urinary Surgery.
Henry F. Graff, A.B., M.D., Associate in Ophthalmology.
Harold M. Goodman, A.B., M.D., Associate in Dermatology.
Wetherbee Fort, M.D., Associate in Medicine.
F. Stratner Orem, M.D., Associate in Pediatrics.
Frank J. Geraghty, A.B., M.D., Associate in Medicine.
L. P. Gundry, A.B., M.D., Associate in Medicine.
John F. Hogan, M.D., Associate in Genito-Urinary Surgery.
Thomas R. O'Rourk, M.D., Associate in Diseases of the Nose and Throat and Otology and Assistant in Ophthalmology.
Kenneth B. Boyd, M.D., Associate in Gynecology and Assistant in Obstetrics.
John T. Hibbits, M.D., Associate in Gynecology.
E. H. Tonolla, M.D., Associate in Medicine.
C. W. Peake, M.D., Associate in Surgery.
W. A. H. Councill, M.D., Associate in Genito-Urinary Surgery.
Z. Vance Hooper, M.D., Associate in Gastro-Enterology.
Frank B. Kindell, A.B., M.D., Associate in Pathology.
William M. Seabold, A.B., M.D., Associate in Pediatrics.
‡H. Alvan Jones, M.D., Associate in Orthopaedic Surgery.
†Benjamin S. Rich, A.B., M.D., Associate in Otology and in †Diseases of the Nose and Throat.
John E. Savage, B.S., M.D., Associate in Obstetrics and Assistant in Pathology.
‡Joseph C. Bernstein, M.D., Associate in Dermatology.
Thomas J. Coonan, A.B., M.D., Associate in Pediatrics.
E. Eugene Covington, M.D., Associate in Gross Anatomy and Instructor in Surgery.
Edward A. Kitlowski, M.D., Associate in Plastic Surgery.
*G. Bowers Mansdorfer, B.S., M.D., Associate in Pediatrics.
*Sol Smith, M.D., Associate in Medicine.
*Henry V. Langeluttig, A.B., M.D., Associate in Medicine.
*James W. Nelson, M.D., Associate in Surgery.
*I. Ridgeway Trimble, M.D., Associate in Surgery.
*Richard T. Shackelford, A.B., M.D., Associate in Surgery.
*Simon H. Brager, M.D., Associate in Surgery.
*Raymond F. Helfrich, A.B., M.D., Associate in Surgery.
★*Harry S. Shelley, B.S., M.D., Associate in Genito-Urinary Surgery.
*Harry C. Hull, M.D., *Associate in Surgery, Assistant in Pathology.
*Harry M. Robinson, Jr., B.S., M.D., *Associate in Dermatology, Assistant in Medicine.
*Otto C. Brantigan, B.S., M.D., Associate in Surgery and Gross Anatomy.
*Francis W. Gillis, M.D., Associate in Genito-Urinary Surgery.
*John C. Dumler, B.S., M.D., Associate in Gynecology.
*Beverley C. Compton, A.B., M.D., Associate in Gynecology.
*Houston Everett, M.D., Associate in Gynecology.
*Margaret B. Ballard, M.D., Associate in Obstetrics.

*Dudley P. Bowe, A.B., M.D., Associate in Obstetrics.
*Cleo D. Stiles M.D., *Associate in Diseases of the Nose and Throat, and Assistant in Otology.

INSTRUCTORS

★Henry F. Buettner, M.D., Instructor in Bacteriology.
‡J. A. F. Pfeiffer, Ph.D., M.D., Instructor in Bacteriology.
M. S. Koppelman, M.D., Instructor in Gastro-Enterology.
Myron G. Tull, A.B., M.D., Instructor in Hygiene and Public Health.
William R. Johnson, M.D., Instructor in Surgery.
E. M. Hanrahan, A.B., M.D., Instructor in Surgery.
Ruth Musser, M.S., Instructor in Pharmacology.
Benjamin Abeshouse, Ph.B., M.D., Instructor in Pathology.
‡Joseph Pokorney, M.D., Instructor in Histology.
S. Demarco, M.D., Instructor in Surgery.
★Karl J. Steinmueller, A.B., M.D., Instructor in Surgery.
Samuel T. Helms, B.S., M.D., Instructor in Medicine and Genito-Urinary Surgery.
F. A. Holden, M.D., Instructor in Ophthalmology.
§Martin J. Hanna, M.D., Instructor in Histology.
Earl L. Chambers, M.D., Instructor in Medicine.
M. Paul Byerly, M.D., Instructor in Pediatrics.
William E. Hahn, D.D.S., Instructor in Oral Surgery.
Luther E. Little, M.D., Instructor in Surgery.
J. Frank Hewitt, A.B., M.D., Instructor in Surgery.
David Tenner, M.D., Instructor in Medicine.
R. B. Mitchell, Jr., B.S., M.D., Instructor in Medicine.
Samuel Legum, A.B., M.D., Instructor in Medicine.
Ernest Levi, M.D., Instructor in Gastro-Enterology.
Ernest S. Edlow, A.B., M.D., Instructor in Gynecology.
Thomas S. Bowyer, A.B., M.D., Instructor in Gynecology.
Joseph V. Castagna, M.D., Instructor in Gynecology.
M. S. Shiling, A.B., M.D., D.Sc., Instructor in Medicine.
Robert W. Garis, A.B., M.D., Instructor in Medicine.
Philip F. Lerner, A.B., M.D., Instructor in Neurology.
H. Edmund Levin, B.S., M.D., Instructor in Medicine and Bacteriology.
★Conrad B. Acton, B.S., M.D., Instructor in Medicine and Assistant in Pathology.
Robert A. Reiter, A.B., M.D., Instructor in Medicine.
W. Grafton Hersperger, A.B., M.D., Instructor in Medicine.
Meyer W. Jacobson, M.D., Instructor in Medicine.
★Hugh G. Whitehead, M.D., Instructor in Medicine.
F. Edwin Knowles, Jr., M.D., Instructor in Ophthalmology.
Philip D. Flynn, M.D., Instructor in Medicine.
Albert J. Shochat, B.S., M.D., Instructor in Gastro-Enterology.
‡Thomas A. Christensen, B.S., M.D., Instructor in Pediatrics.
Jerome Fineman, M.D., Instructor in Pediatrics.
Bernhard Badt, M.D., Instructor in Neurology.
William L. Fearing, M.D., Instructor in Neurology.
Frank K. Morris, A.B., M.D., Instructor in Gynecology and Obstetrics.

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§ Died December 2, 1940.

B. Matthew Debuskey, M.D., Instructor in Pediatrics.
★A. Russell Anderson, M.D., Instructor in Psychiatry.
James C. Owings, M.D., Instructor in Diseases of the Rectum and Colon.
Edward S. Kallins, B.S., M.D., Instructor in Medicine.
W. Allen Deckert, A.B., M.D., Instructor in Gynecology and Assistant in Surgery and Obstetrics.
Murray M. Copeland, M.D., Instructor in Surgery.
J. Duer Moores, B.S., M.D., Instructor in Surgery.
★Philip S. Wagner, M.D., Instructor in Psychiatry.
★Irving Freeman, B.S., M.D., Instructor in Medicine.
‡James U. Thompson, A.B., M.D., Instructor in Gross Anatomy.
[3]‡Walter H. Gerwig, Jr., M.D., Instructor in Physiology.
*Hugh B. McNally, B.S., M.D., Instructor in Obstetrics.
*John A. Myers, M.E.E., M.D., *Instructor in Medicine, Assistant in Gastro-Enterology.
†Edward F. Cotter, M.D., †Instructor in Pathology, Assistant in Neurology.
*Kurt Levy, M.D., Instructor in Medicine.
*Thurston R. Adams, M.D., Instructor in Surgery.
*†Henry L. Wollenweber, M.D., *Instructor in Pathology and †Assistant in Pharmacology.
*William G. Helfrich, B.S., M.D., Instructor in Medicine.
*†Howard B. Mays, M.D., *Instructor in Genito-Urinary Surgery and †Assistant in Pathology.
*Arthur G. Siwinski, A.B., M.D., Instructor in Surgery.
★*Lester N. Kolman, M.D., Instructor in Dermatology.
*Gibson J. Wells, A.B., M.D., Instructor in Pediatrics.
†Harry A. Teitelbaum, B.S., M.D., †Instructor in Gross Anatomy, Assistant in Neurology.
†Ernest I. Cornbrooks, Jr., M.D., Instructor in Gynecology.
†Francis G. Dickey, A.B., M.D., Instructor in Medicine.
†Herbert E. Reifschneider, A.B., M.D., Instructor in Oral Surgery and Assistant in Surgery.
†William B. Settle, M.D., Instructor in Gynecology.
†Robert L. Graham, A.B., M.D., Instructor in Pathology.
†Conrad L. Inman, D.D.S., Instructor in Oral Surgery.
†Vernon D. Kaufman, D.D.S., Instructor in Oral Surgery.
†B. Wallace Inman, D.D.S., Instructor in Oral Surgery.
†Dorsey R. Tipton, D.D.S., Instructor in Oral Surgery.
†I. H. Maseritz, M.D., Instructor in Orthopaedic Surgery.

ASSISTANTS

William R. Geraghty, B.S., M.D., Assistant in Surgery.
Jaroslav Hulla, M.D., Assistant in Obstetrics.
Marius P. Johnson, A.B., M.D., Assistant in Pharmacology and Obstetrics.
Howard B. McElwain, M.D., Assistant in Surgery.
A. V. Buchness, A.B., M.D., Assistant in Surgery.
T. J. Touhey, M.D., Assistant in Surgery.
W. J. Schmitz, M.D., Assistant in Pediatrics.

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[3] Military service.

JOHN G. RUNKLE, M.D., Assistant in Ophthalmology.
CLYDE F. KARNS, B.S., M.D., Assistant in Surgery.
MORRIS FINE, M.D., Assistant in Medicine.
SAMUEL H. CULVER, M.D., Assistant in Surgery.
★W. H. TRIPLETT, M.D., Assistant in Medicine.
E. HOLLISTER DAVIS, A.B., M.D., Assistant in Anaesthesia.
ISRAEL P. MERANSKI, B.S., M.D., Assistant in Pediatrics.
ALBERT R. WILKERSON, M.D., Assistant in Surgery.
‡DOROTHY E. SCHMALZER, B.S., Assistant in Biological Chemistry.
H. WHITNEY WHEATON, M.D., Assistant in Pediatrics.
LAURISTON L. KEOWN, A.B., M.D., Assistant in Pediatrics.
L. T. CHANCE, M.D., Assistant in Surgery.
SAMUEL McLANAHAN, JR., A.B., M.D., Assistant in Surgery.
★SAMUEL E. PROCTOR, A.B., M.D., Assistant in Surgery.
GEORGE GOVATOS, A.B., M.D., Assistant in Surgery.
JEROME SNYDER, B.S., M.D., Assistant in Ophthalmology.
WILLIAM H. GRENZER, M.D., Assistant in Medicine.
GEORGE SILVERTON, A.B., M.D., Assistant in Medicine.
DWIGHT M. CURRIE, A.B., M.D., Assistant in Surgery.
ROLLIN C. HUDSON, M.D., Assistant in Dermatology.
‡THOMAS E. ROACH, B.S., M.D., Assistant in Dermatology.
MILTON C. LANG, M.D., Assistant in Ophthalmology.
MAXWELL L. MAZER, M.D., Assistant in Obstetrics.
★JOSEPH M. BLUMBERG, B.S., M.D., Assistant in Medicine and Obstetrics.
FERD. E. KADAN, A.B., M.D., Assistant in Obstetrics.
LAWRENCE KATZENSTEIN, M.D., Assistant in Medicine.
LEON ASHMAN, M.D., Assistant in Medicine.
NATHANIEL M. BECK, A.B., M.D., Assistant in Medicine and Gastro-Enterology.
‡ABRAHAM HURWITZ, M.D., Assistant in Pediatrics.
CHARLES E. BALFOUR, M.D., Assistant in Neurology.
ROBERT F. HEALY, M.D., Assistant in Surgery.
HERBERT M. FOSTER, M.D., Assistant in Surgery.
DANIEL R. ROBINSON, M.D., Assistant in Surgery.
H. L. GRANOFF, A.B., M.D., Assistant in Gynecology.
J. J. ERWIN, M.D., Assistant in Gynecology.
ISRAEL ROSEN, A.B., M.D., Assistant in Pediatrics.
WILLIAM GREENFELD, M.D., Assistant in Gastro-Enterology.
S. EDWIN MULLER, M.D., Assistant in Medicine.
SAMUEL SNYDER, M.D., Assistant in Medicine.
PAUL E. CARLINER, B.S., M.D., Assistant in Medicine.
WILLIAM A. PARR, M.D., Assistant in Otology.
JEANNETTE R. HEGHINIAN, B.S., M.D., Assistant in Dermatology.
JOSEPH V. JERARDI, B.S., M.D., Assistant in Surgery.
ROBERT F. CHENOWITH, M.D., Assistant in Surgery.
JACOB R. JENSEN, B.S., M.D., Assistant in Obstetrics.
J. EDWARD NORRIS, M.D., Assistant in Obstetrics.
★J. WARREN ALBRITTAIN, B.S., M.D., Assistant in Obstetrics.
†KARL F. MECH, B.S., M.D., Assistant in Pathology.
JOYE E. JACOBS, A.B., Assistant in Physiology.
LOUIS J. KROLL, A.B., M.D., Assistant in Medicine.
†‡SAMUEL T. R. REVELL, B.S., M.D., †Assistant in Medicine and ‡Assistant in Pathology.

Hugh Jewett, M.D., Assistant in Genito-Urinary Surgery.
John V. Hopkins, M.D., Assistant in Orthopaedic Surgery.
Eldred Roberts, B.S., M.D., Assistant in Surgery.
‡Alexander McGill Duff, M.D., Assistant in Surgery.
John L. Atkins, A.B., M.D., Assistant in Medicine.
John W. Machen, M.D., Assistant in Neurology.
[3]‡Arnold L. Peter, M.D., Assistant in Ophthalmology.
Donald B. Grove, M.D., Assistant in Surgery.
William S. Cheney, M.D., Assistant in Surgery.
†George H. Davis, M.D., Assistant in Obstetrics.
†F. A. Kayser, M.D., Assistant in Diseases of the Nose and Throat.
†Theodore A. Schwartz, M.D., Assistant in Diseases of the Nose and Throat.
†Harry C. Bowie, B.S., M.D., Assistant in Surgery and Gross Anatomy.
†D. McClellen Dixon, M.D., Assistant in Obstetrics.
†Ephraim T. Lisansky, A.B., M.D., Assistant in Pathology and Medicine.
†William K. Waller, A.B., M.D., Assistant in Medicine.
†Robert J. Gore, M.D., Assistant in Pediatrics.
†Earle S. Scott, M.D., Assistant in Pediatrics.
†Nachman Davidson, A.B., M.D., Assistant in Medicine.
†William M. Garlick, A.B., M.D., Assistant in Surgery.
†Ann Virginia Brown, A.B., Assistant in Biological Chemistry.
†George H. Brouillet, B.S., M.D., Assistant in Surgery.
†Benjamin Highstein, M.D., Assistant in Dermatology.
†Mark Hollander, M.D., Assistant in Dermatology.
†David Hollander, A.B., M.D., Assistant in Pathology.
★†W. Kenneth Mansfield, Jr., M.D., Assistant in Obstetrics.
†Bernard W. Donahue, M.D., Assistant in Obstetrics.
†C. Edward Leach, M.D., Assistant in Medicine.
†Richard N. Tillman, M.D., Assistant in Medicine.

FELLOWS

Frederick K. Bell, Ph.D., U. S. Pharmacopoeia Fellow.
Sylvan E. Forman, Ph.D., Fellow in Pharmacology.
Fred W. Ellis, Ph.D., Fellow in Pharmacology.
Wilson C. Grant, M.S., International Cancer Research Foundation, Research Assistant in Pharmacology.
Sidney Berman, M.D., Fellow in Psychiatry.
‡Arthur G. Siwinski, A.B., M.D., Fellow National Cancer Institute.
‡Alexander McGill Duff, M.D., Fellow National Cancer Institute.
‡William K. Waller, A.B., M.D., Clinical Fellow in Medicine.
‡Otto C. Brantigan, B.S., M.D., Weaver Fellow in Gross Anatomy.
‡Gordon M. Stephens, A.B., M.D., Fellow in Psychiatry.
‡Edward F. Cotter, M.D., Hitchcock Fellow in Neuro-Surgery.
‡Ephraim T. Lisansky, A.B., M.D., Weaver Fellow in Pathology.
‡Nellie Cone, A.B., Emerson Fellow in Pharmacology.
‡Edward Merdinyan, M.S., Emerson Fellow in Pharmacology.
‡Howard B. Mays, M.D., Cranberry Canners, Inc., Fellow in Urology and Biological Chemistry.

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[3] Military service.

‡Ann Virginia Brown, A.B., Cranberry Canners, Inc., Research Assistant in Biological Chemistry.

‡Charles W. Reavis, M.D., Fellow in Roentgenology.

‡†Edward G. Boettiger, Ph.D., †Weaver Fellow in Gynecology, ‡Hitchcock Fellow in Gynecology.

†Frederick J. Vollmer, B.S., M.D., Hitchcock Fellow in Medicine.

†John A. Wagner, B.S., M.D., Weaver Fellow in Pathology.

†Harry C. Bowie, B.S., M.D., Weaver Fellow in Gross Anatomy.

†Guy M. Everett, B.S., Weaver Fellow in Physiology.

†Maurice M. Rath, A.B., Emerson Fellow in Pharmacology.

†Norman Pinschmidt, M.S., Fellow in Pharmacology.

†Hans Loewald, M.D., Fellow in Psychiatry.

†Charles H. Davidson, M.D., Fellow in Roentgenology.

UNIVERSITY HOSPITAL COUNCIL

Harry Clifton Byrd, *President.*
H. Boyd Wylie, *Acting Dean of the School of Medicine.*
M. C. Pincoffs, *Head of the Department of Medicine.*
A. M. Shipley, *Head of the Department of Surgery.*
J. Mason Hundley, Jr., *Head of the Department of Gynecology.*
Louis H. Douglass, *Head of the Department of Obstetrics.*
Clyde A. Clapp, *Head of the Department of Ophthalmology.*
John E. Savage, *Acting Superintendent of the Hospital.*
Miss Annie Crighton, R.N., *Superintendent of Nurses.*
Mrs. R. B. Pue, *Representing Woman's Auxiliary Board.*

Ralph P. Truitt Charles A. Reifschneider	*Representing Hospital Staff.*
George F. Sargent Alfred T. Gundry	*Representing Medical Alumni.*

UNIVERSITY HOSPITAL STAFF

John E. Savage *Acting Superintendent*

Physician-in-Chief	Maurice C. Pincoffs
Physicians	§Harry M. Stein Walter A. Baetjer Thomas R. Sprunt John E. Legge Louis A. M. Krause G. Carroll Lockard Joseph E. Gichner William H. Smith William S. Love, Jr. T. Nelson Carey Raymond Hussey
Gastro-Enterologist	Zachariah R. Morgan
Neurologists	Irving J. Spear George M. Settle Leon Freedom
Psychiatrists	Ross McC. Chapman Ralph P. Truitt Harry M. Murdock
Pediatricians	C. Loring Joslin Albert Jaffe J. E. Bradley
Pathologist	Hugh R. Spencer
Surgeon-in-Chief	Arthur M. Shipley
Surgeons	Charles Reid Edwards Page Edmunds Thomas B. Aycock
Neurological Surgeons	Charles Bagley, Jr. Richard G. Coblentz

Laryngologists	Edward A. Looper Franklin B. Anderson Thomas R. O'Rourk
Proctologists	J. Dawson Reeder Monte Edwards
Orthopaedic Surgeons	Allen Fiske Voshell Moses Gellman Compton Riely Henry F. Ullrich
Genito-Urinary Surgeons	W. H. Toulson W. A. H. Councill Lyle J. Millan ★Harry S. Shelley Howard B. Mays
Roentgenologists	‡Henry J. Walton Walter L. Kilby Eugene L. Flippin
Dermatologist	Harry M. Robinson
Bronchoscopist	Edward A. Looper
Otologist	J. W. Downey, Jr.
Anaesthetists	S. Griffith Davis E. Hollister Davis Mary J. O'Brien, R.N. Ruth Elliott, R.N.
Obstetricians	L. H. Douglass M. A. Novey Isadore A. Siegel J. G. M. Reese J. E. Savage
Ophthalmologists	Clyde A. Clapp William Tarun Henry F. Graff
Gynecologists	J. M. Hundley, Jr. R. G. Willse Leo Brady
Oncologists	J. M. Hundley, Jr. Grant E. Ward

ANNUAL HOSPITAL APPOINTMENTS

The following annual appointments are made from among the graduates of the school to the University Hospital:

Seven Residents in Surgery
Four Residents in Medicine
Four Residents in Obstetrics
Two Residents in Gynecology
Three Residents in Pediatrics
Resident in Nose & Throat
Three Residents in Roentgenology
Twelve Senior Internes rotating in Medicine and Surgery
Twelve Junior Internes rotating in the Specialties

UNIVERSITY HOSPITAL RESIDENT AND INTERN STAFF
JULY 1, 1940 TO JUNE 30, 1941

RESIDENT STAFF

Harry C. Bowie, B.S., M.D., *Resident Surgeon.*
Stuart G. Coughlan, B.S., M.D., *First Assistant Resident Surgeon.*
[2]William B. Long, Jr., B.S., M.D., *First Assistant Resident Surgeon.*
John H. Haase, M.D., *Assistant Resident Surgeon.*
Robert C. Sheppard, M.D., *Assistant Resident Surgeon.*
Robert L. Gottschalk, A.B., M.D., *Assistant Resident Surgeon.*
★Bernard Rabold, B.S., M.D., *Assistant Resident Surgeon.*
James G. Arnold, Jr., A.B., M.D., *Assistant Resident Surgeon assigned to Neuro-Surgery.*
William C. Humphries, A.B., M.D., *Assistant Resident Surgeon assigned to Eye, Ear, Nose and Throat.*
★Stephen L. Magness, A.B., M.D., *Surgical Intern.*
W. H. Fisher, M.D., *Surgical Intern.*
John L. Atkins, A.B., M.D., *Resident Physician.*
★John Z. Bowers, B.S., M.D., *Assistant Resident Physician.*
★Timothy A. Callahan, Jr., M.D., *Assistant Resident Physician.*
Conover Talbot, A.B., M.D., *Assistant Resident Physician.*
Glenn H. Algire, B.S., M.D., *Medical Intern.*
John K. B. E. Seegar, Jr., A.B., M.D., *Resident Obstetrician.*
M. M. Schneider, M.D., *Assistant Resident Obstetrician.*
Louis C. Gareis, M.D., *Assistant Resident Obstetrician.*
Robert Linn, M.D., *Assistant Resident Obstetrician.*
Raphael A. Vilar, M.D., *Obstetrical Intern.*
Everett S. Diggs, B.S., M.D., *Resident Gynecologist.*
William K. Diehl, B.S., M.D., *Assistant Resident Gynecologist.*
Roland E. Bieren, M.D., *Assistant Resident Gynecologist.*
Charles N. Davidson, M.D., *Resident Roentgenologist.*
George W. McCall, A.B., M.D., *Assistant Resident Roentgenologist.*
Charles W. Reavis, M.D., *Fellow in Roentgenology.*
Robert J. Gore, A.B., M.D., *Resident Pediatrician.*
Earl S. Scott, M.D., *Assistant Resident Pediatrician.*
Elizabeth Bess Cannon, A.B., M.D., *Assistant Resident Pediatrician.*

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[2] On leave, 1941–1942.

SENIOR INTERNS

Henry A. Briele, M.D.
Raymond M. Cunningham, A.B., M.D.
Leo J. Gaver, M.D.
Alvin S. Hartz, A.B., M.D.
William H. Leitch, B.S., M.D.
Francis J. McLaughlin, M.D.
William S. Miller, A.B., M.D.
★James B. Nuttall, M.D.
Leslie H. Pierce, B.S., M.D.
[2]Edwin R. Ruzicka, B.S., M.D.
Leland B. Stevens, B.S., M.D.
★Ramsay B. Thomas, B.S., M.D.

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[2] On leave, 1941–1942.

JUNIOR INTERNS

Lester H. Caplan, A.B., M.D.
Edwin O. Daue, B.S., M.D.
R. L. Gibbs, Jr., A.B., M.D.
Walter R. Graham, A.B., M.D.
Benjamin H. Inloes, Jr., M.D.
James R. Karns, M.D.
H. Pearce Maccubbin, B.S., M.D.
Robert B. Mearns, M.D.
C. Martin Rhode, A.B., M.D.
Alexander F. Thompson, A.B., M.D.
Wilfred H. Townshend, Jr., A.B., M.D.
Richard T. Williams, A.B., M.D.

DENTAL INTERN

Samuel Goldhaber, D.D.S.

ANNOUNCEMENT OF UNIVERSITY HOSPITAL RESIDENT AND INTERN STAFF EFFECTIVE JULY 1, 1941

RESIDENT STAFF

Stuart G. Coughlan, B.S., M.D., *Resident Surgeon.*
John H. Haase, M.D., *Assistant Resident Surgeon.*
Robert C. Sheppard, M.D., *Assistant Resident Surgeon.*
Henry A. Briele, M.D., *Assistant Resident Surgeon.*
Raymond Cunningham, A.B., M.D., *Assistant Resident Surgeon.*
Philip L. Smoak, M.D., *Assistant Resident Surgeon.*
James G. Arnold, Jr., A.B., M.D., *Assistant Resident Surgeon assigned to Neuro-Surgery.*
Lawrence S. Cannon, A.B., M.D., *Assistant Resident Surgeon assigned to Neuro-Surgery.*
Richard S. Owens, Jr., M.D., *Assistant Resident Surgeon assigned to Eye, Ear, Nose and Throat.*
William H. Fisher, M.D., *Surgical Intern.*
Forest C. Meade, M.D., *Surgical Intern.*
Samuel T. R. Revell, Jr., B.S., M.D., *Resident Physician.*
Leslie H. Pierce, B.S., M.D., *Assistant Resident Physician.*
Alvin S. Hartz, A.B., M.D., *Assistant Resident Physician.*
Robert Linn, M.D., *Resident Obstetrician.*
William S. Miller, A.B., M.D., *Assistant Resident Obstetrician.*
Richard L. Goyne, B.S., M.D., *Assistant Resident Obstetrician.*
Martin Clyman, B.S., M.D., *Assistant Resident Obstetrician.*
John T. Cole, M.D., *Obstetrical Intern.*
William K. Diehl, B.S., M.D., *Resident Gynecologist.*
Roland E. Bieren, M.D., *Assistant Resident Gynecologist*
Edward G. Jones, A.B., M.D., *Assistant Resident Gynecologist.*
Charles N. Davidson, M.D., *Fellow in Roentgenology.*
George W. McCall, A.B., M.D., *Resident Roentgenologist.*

James J. McCort, A.B., M.D., *Assistant Resident Roentgenologist.*
Earl S. Scott, A.B., M.D., *Resident Pediatrician.*
Arnold F. Lavenstein, A.B., M.D., *Assistant Resident Pediatrician.*

SENIOR INTERNS

Lester H. Caplan, A.B., M.D.
Edwin O. Daue, B.S., M.D.
R. L. Gibbs, Jr., A.B., M.D.
Walter R. Graham, A.B., M.D.
Benjamin H. Inloes, Jr., M.D.
James R. Karns, M.D.
H. Pearce Maccubbin, B.S., M.D.
Robert B. Mearns, M.D.
C. Martin Rhode, A.B., M.D.
Alexander F. Thompson, A.B., M.D.
Wilfred H. Townshend, Jr., A.B., M.D.
Richard T. Williams, A.B., M.D.

JUNIOR INTERNS

Joshua W. Baxley, III, B.S., M.D.
James S. Hunter, Jr., M.D.
Norval Kemp, A.B., M.D.
Lorman L. Levinson, A.B., M.D.
Margaret V. Palmer, M.A., M.D.
Walter J. Revell, B.S., M.D.
Edwin L. Seigman, A.B., M.D.
Webster M. Strayer, A.B., M.D.
Raymond K. Thompson, B.S., M.D.
George J. Ulrich, A.B., M.D.
James H. Walker, A.B., M.D.
Thomas C. Wilder, M.D.

DENTAL INTERN

Edward P. McDaniel, Jr., D.D.S.

UNIVERSITY HOSPITAL DISPENSARY STAFF

Emma Winship, R.N.
Dispensary Director

DISPENSARY COMMITTEE

John E. Legge, *Chairman*
Emma Winship, R.N., *Secretary*

L. H. Douglass
J. Mason Hundley, Jr.
C. Loring Joslin
John E. Savage
George H. Yeager
Ralph P. Truitt

Chief of Medical Clinic....................................John E. Legge

Assistant Chiefs of Medical Clinic........................
Lewis P. Gundry
★Irving Freeman
*Kurt Levy

Physicians..
Harry G. Jones
M. A. Fine
★Herbert Goldstone
‡Charles H. Reier
‡Nachman Davidson
‡George Silverton
‡Carl P. Roetling
‡Albert J. Himelfarb

Chief of Gastro-Enterology Clinic..........................Zachariah Morgan

Assistant Chief of Gastro-Enterology Clinic	†Francis G. Dickey
Assistant Gastro-Enterologists	Z. V. Hooper Ernest Levi ‡John A. Myers M. S. Koppelman Samuel Morrison Albert J. Shochat William Greenfeld
Chief of Neurology Clinic	Leon Freedom
Assistant Neurologists	Benjamin Pushkin Bernhard Badt William L. Fearing ‡Charles E. Balfour Harry A. Teitelbaum
Chief of Psychiatric Clinic	Ralph P. Truitt
Assistant Psychiatrists	H. W. Newell ★Harry Goldsmith ‡Harry Murdock Alice J. Rockwell ‡Gordon M. Stephens †Joseph Marshall †H. Robert Blank †William W. Elgin †Samuel Barkoff †Hans Loewald †Sidney Berman
Chief of Chest Clinic	E. H. Tonolla
Assistants, Diseases of the Lungs	‡H. V. Langeluttig Manuel Levin Meyer W. Jacobson John Constantini †Carl P. Roetling †Joseph E. Muse, Jr.
Chief of Metabolism Clinic	Lewis P. Gundry
Assistants in Metabolism Clinic	William G. Helfrich Walter E. Karfgin A. Goldman
Chief of Cardiovascular Clinic	William S. Love, Jr.
Assistant Chiefs of Cardiovascular Clinic	Robert W. Garis Robert B. Mitchell, Jr.

Assistant Cardiologists	Samuel J. Hankin Anthony F. Carozza W. G. Hersperger Richard S. Owens ★Irving Freeman Harry M. Robinson, Jr. †Ephraim T. Lisansky †C. Edward Leach
Chief of Allergy Clinic	H. M. Bubert
Assistant Chiefs of Allergy Clinic	Edward S. Kallins ‡Richard S. Owens
Assistant Allergists	Paul E. Carliner Nachman Davidson Samuel Snyder Benjamin S. Rich Frank V. Manieri ★‡Irving Freeman †S. Edwin Muller †Richard N. Tillman
Allergy Clinic Technician	Jane Wood
Director, Pediatric Clinic	C. Loring Joslin
Chief of Pediatric Clinic	John H. Traband
Chief of Pediatric Clinic	Clewell Howell
Assistant Pediatricians	M. Paul Byerly A. H. Finkelstein Samuel S. Glick F. Strattner Orem Israel P. Meranski ‡Robert B. Taylor William M. Seabold Jerome Fineman H. Whitney Wheaton Albert Jaffe S. Lilienfeld ‡Thomas A. Christensen Lauriston L. Keown Israel Rosen ‡Abraham B. Hurwitz Joseph R. Meyerowitz ‡Louis R. Maser Gibson J. Wells Robert J. Gore ‡Elizabeth Bess Cannon †Louis B. Blum †Earl S. Scott †Arnold F. Lavenstein

Chief of Surgical Clinic	George H. Yeager
Assistant Surgeons	J. Frank Hewitt Thurston R. Adams Luther E. Little George H. Brouillet W. R. Johnson ‡Karl F. Mech ★Samuel E. Proctor C. Vernon Williamson Albert R. Wilkerson Robert Chenowith
Chief of Orthopaedic Surgery Clinic	Allen Fiske Voshell
Assistant Orthopaedic Surgeons	John V. Hopkins Moses Gellman Henry F. Ullrich Fred Duckwall
Chief of Genito-Urinary Clinic	W. H. Toulson
Assistant Genito-Urinary Surgeons	W. A. H. Councill John F. Hogan L. J. Millan ★Harry S. Shelley Howard B. Mays
Chief of Roentgenology Clinic	‡Henry J. Walton Walter L. Kilby
Assistant Roentgenologist	Eugene L. Flippin
Chief of Dermatology Clinic	Harry M. Robinson
Assistant Dermatologists	Rollin C. Hudson Harold M. Goodman Harry M. Robinson, Jr. Jeannette R. Heghinian Lester N. Kolman †Mark Hollander †Benjamin Highstein
Chief of Rhinology and Laryngology Clinic	‡Franklin B. Anderson *Benjamin S. Rich
Assistant Rhinologists and Laryngologists	Joseph Nurkin Cleo D. Stiles Thomas R. O'Rourk M. Baylus
Chief of Proctology Clinic	Monte Edwards
Assistant Proctologist	James C. Owings
Chief of Gynecology Clinic	J. Mason Hundley, Jr.
Assistant Chief of Gynecology Clinic	Beverley C. Compton

Assistant Gynecologists	Thomas S. Bowyer Joseph V. Castagna John C. Dumler John T. Hibbitts ‡Hugh B. McNally †Ernest I. Cornbrooks, Jr. †William B. Settle Kenneth B. Boyd W. Allen Deckert Marius P. Johnson Everett S. Diggs Charles B. Marek Helen I. Maginnis Irvin P. Klemkowski
Female Cystoscopists	J. Mason Hundley, Jr. W. Allen Deckert Beverley C. Compton John C. Dumler
Chief of Oral Surgery Clinic	Brice M. Dorsey
Oral Surgeons	Harold Goldstein Samuel H. Bryant
Chief of Obstetrical Clinic	John E. Savage
Assistant Obstetricians	Margaret B. Ballard Maxwell L. Mazer Jaroslav Hulla Marius P. Johnson Dudley P. Bowe W. Allen Deckert ★Joseph M. Blumberg Hugh B. McNally Ferd E. Kadan Jacob R. Jensen J. Edward Norris ★J. Warren Albrittain
Oncology Clinic, Gynecological Division	J. Mason Hundley, Jr. Thomas S. Bowyer Beverley C. Compton John C. Dumler
Oncology Clinic, Surgical Division	Grant E. Ward E. Eugene Covington J. Duer Moores J. W. Nelson ‡Eldred Roberts Murray M. Copeland ‡Alexander McHill Duff Arthur G. Siwinski

Chief of Ophthalmology Clinic	H. F. Graff
Assistant Ophthalmologists	Frank A. Holden Thomas R. O'Rourk Milton C. Lang John G. Runkle Jerome Snyder [3]‡Arnold L. Peter
Directress, Occupational Therapy	Miss Nancy Valliant
Directress, Social Service	Miss Grace Pearson

UNIVERSITY HOSPITAL DISPENSARY REPORT

October 1, 1939 to September 30, 1940

Departments	*New Cases*	*Old Cases*	*Total*
Allergy Clinic	183	5,777	5,960
Cardiology	283	2,421	2,704
Curative Workshop	35	1,016	1,051
Cystoscopy	133	495	628
Dermatology	4,554	16,900	21,454
Diabetic	73	1,240	1,313
Gastro-Intestinal	180	1,085	1,265
Genito-Urinary	700	2,695	3,395
Gynecology	1,954	5,345	7,299
Medicine	2,324	5,036	7,360
Neurology	261	1,087	1,348
Nose, Throat and Ear	1,507	1,305	2,812
Obstetrics	1,759	10,275	12,034
Oncology	396	2,488	2,884
Ophthalmology	1,619	4,247	5,866
Oral Surgery	702	863	1,565
Orthopaedic	1,712	4,320	6,032
Pediatrics	2,601	12,768	15,369
Physiotherapy	227	2,210	2,437
Proctology	254	280	534
Psychiatry	59	297	356
Surgery	3,141	9,128	12,269
Tuberculosis	289	2,745	3,034
Total	24,946	94,023	118,969

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[3] Military service.

MERCY HOSPITAL

BOARD OF GOVERNORS

Walter D. Wise, *Chairman*

Sister M. Veronica	Henry F. Bongardt
Sister M. Celeste	Maurice C. Pincoffs
Sister M. Hildegarde	Waitman F. Zinn
Sister M. de Chantal	Thomas K. Galvin
Sister M. Hilda	Edward P. Smith

Sister M. Joseph

MERCY HOSPITAL STAFF

Consulting Surgeon	§Alexius McGlannan
Surgeon-in-Chief	Walter D. Wise
Surgeons	Charles F. Blake F. L. Jennings R. W. Locher Thomas R. Chambers Elliott H. Hutchins D. J. Pessagno William F. Rienhoff N. Clyde Marvel H. F. Bongardt
Associate Surgeons	I. O. Ridgely J. W. Nelson Howard B. McElwain Simon H. Brager John A. O'Connor Thomas B. Aycock Charles W. Maxson I. Ridgeway Trimble Richard T. Shackelford Raymond F. Helfrich
Assistant Surgeons	Julius Goodman E. Eugene Covington S. DeMarco, Jr. T. J. Touhey William N. McFaul, Jr. Meyer H. Zuravin ★Howard L. Zupnik Daniel R. Robinson Joseph V. Jerardi
Consulting Ophthalmologist and Otologist	Harry Friedenwald
Ophthalmologists and Otologists	H. K. Fleck J. W. Downey M. Raskin

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§ Died, February 25, 1940.

Associate Ophthalmologists and Otologists	Joseph I. Kemler F. A. Pacienza
Assistant Ophthalmologist and Otologist	★Joseph V. Jeppi
Consulting Rhinologists and Laryngologists	W. Raymond McKenzie George W. Mitchell
Rhinologist and Laryngologist	Waitman F. Zinn
Associate Rhinologists and Laryngologists	F. A. Pacienza F. A. Kayser Benjamin S. Rich Theodore A. Schwartz Birkhead Macgowan
Assistant Rhinologist and Laryngologist	★Joseph V. Jeppi
Bronchoscopist	Waitman F. Zinn
Associate Bronchoscopist	F. A. Kayser
Assistant Bronchoscopist	Theodore A. Schwartz
Proctologist	Charles F. Blake
Assistant Proctologist	E. Eugene Covington
Orthopaedic Surgeon	Albertus Cotton
Associate Orthopaedic Surgeon	H. L. Rogers
Assistant Orthopaedic Surgeons	I. H. Maseritz J. H. Gaskel
Urologist	Kenneth D. Legge
Associate Urologists	Leon K. Fargo Francis W. Gillis
Dentist	J. D. Fusco
Assistant Dentists	J. J. Foley B. Wallace Inman
Physician-in-Chief	Maurice C. Pincoffs
Physicians	C. B. Gamble Harvey G. Beck Thomas P. Sprunt H. Raymond Peters George McLean J. Sheldon Eastland Louis A. M. Krause
Associate Physicians	Hubert C. Knapp Bartus T. Baggott Thomas C. Wolff Wetherbee Fort John E. Legge T. Nelson Carey
Assistant Physicians	Sol Smith S. A. Tumminello J. Howard Burns Earl L. Chambers K. W. Golley J. M. Miller

Gastro-Enterologist	§Julius Friedenwald
Associate Gastro-Enterologists	T. Frederick Leitz Theodore H. Morrison
Assistant Gastro-Enterologists	Maurice Feldman Joseph Sindler
Pediatricians	Edgar B. Friedenwald Frederick B. Smith
Associate Pediatricians	Thomas J. Coonan G. Bowers Mansdorfer
Assistant Pediatricians	William J. Schmitz William M. Seabold
Neurologist and Psychiatrist	Andrew C. Gillis
Associate Neurologists and Psychiatrists	★Harry Goldsmith Philip F. Lerner
Obstetricians	Edward P. Smith Abram S. Samuels Ernest S. Edlow William S. Gardner Thomas K. Galvin John J. Erwin
Associate Obstetrician	‡Frank K. Morris †Harold H. Burns
Assistant Obstetrician	William B. Smith
Consulting Gynecologist	William S. Gardner
Gynecologists	Abram S. Samuels Thomas K. Galvin Edward P. Smith George S. Strauss, Jr.
Associate Gynecologists	J. J. Erwin Ernest S. Edlow Frank K. Morris
Assistant Gynecologist	Harry L. Granoff
Pathologists	Walter C. Merkel Hugh R. Spencer
Clinical Pathologists	H. T. Collenberg H. R. Peters Charles E. Bramble
Technicians	Eleanor Behr Anna Wassell Audrey Auten
Radiographer	Albertus Cotton
Associate Radiographer	Harry L. Rogers
Assistant Radiographer	†A. D. Young ‡Harry Archer Miller
Technicians	Sister M. Kevin Charlotte Clopter

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§ Deceased, June 8, 1941.

ANNUAL HOSPITAL APPOINTMENTS

The following annual appointments are made from among the graduates of the school to the Mercy Hospital:

Five Residents in Surgery
Two Residents in Medicine
Resident in Rhinology
Two Residents in Gynecology
Ten Interns on Rotating Service

MERCY HOSPITAL RESIDENT AND INTERN STAFF
JULY 1, 1940 TO JUNE 30, 1941

RESIDENT STAFF

W. Lynn Garlick, A.B., M.D., *Resident Surgeon.*
John F. Schaefer, B.S., M.D., *Associate Resident Surgeon.*
Howard F. Kinnamon, Jr., M.D., *Assistant Resident Surgeon.*
Richard W. Corbitt, A.B., M.D., *Assistant Resident Surgeon.*
Melvin F. Polek, B.S., M.D., *Assistant Resident Surgeon.*
Frederick J. Vollmer, B.S., M.D., *Resident Physician.*
William H. Kammer, Jr., A.B., M.D., *Assistant Resident Physician.*
John A. Wagner, B.S., M.D., *Assistant Resident Physician.*
★Joseph E. Schenthal, A.B., M.D., *Assistant Resident Physician and Resident Pathologist.*
Charles H. Doeller, Jr., A.B., M.D., *Resident Gynecologist.*
William A. Dodd, B.S., M.D., *Resident Obstetrician.*
Harry McBrine Beck, A.B., M.D., *Assistant Resident Gynecologist and Obstetrician.*
Maynard C. Smith, B.S., M.D., *Resident Rhinologist.*

INTERNS

George B. Barnes, M.D.
Paul H. Correll, A.B., M.D.
William C. Duffy, A.B., M.D.
James A. Freeman, M.D.
William C. Livingood, B.S., M.D.
F. Ford Loker, B.S., M.D.
William D. McClung, M.D.
Forest C. Meade, M.D.
Donald J. Roop, A.B., M.D.
William E. Weeks, B.S., M.D.

ANNOUNCEMENT OF
MERCY HOSPITAL RESIDENT AND INTERN STAFF

EFFECTIVE JULY 1, 1941

RESIDENT STAFF

John F. Schaefer, B.S., M.D., *Resident Surgeon.*
Howard F. Kinnamon, Jr., M.D., *Associate Resident Surgeon.*
F. Ford Loker, B.S., M.D., *Assistant Resident Surgeon.*
George B. Barnes, M.D., *Assistant Resident Surgeon.*
Melvin F. Polek, B.S., M.D., *Assistant Resident Surgeon.*
John L. Gillespie, B.S., M.D., *Special Assistant Surgical Resident.*
William H. Kammer, Jr., A.B., M.D., *Resident Physician.*
Donald J. Roop, A.B., M.D., *Assistant Resident Physician.*
Raymond V. Robinson, M.D., *Assistant Resident Physician.*
William E. Weeks, B.S., M.D., *Assistant Resident Physician.*
William A. Dodd, B.S., M.D., *Resident Gynecologist.*
Harry McBrine Beck, A.B., M.D., *Resident Obstetrician.*

Paul H. Correll, A.B., M.D., *Assistant Resident in Gynecology and Obstetrics.*
William C. Duffy, A.B., M.D., *Assistant Resident in Gynecology and Obstetrics.*
William C. Livingood, B.S., M.D., *Resident Otolaryngologist.*

INTERNS

Charles P. Barnett, A.B., M.D.
Michael L. DeVincentis, B.S., M.D.
Edward L. Frey, Jr., A.B., M.D.
Thomas A. Hedrick, M.D.
William C. Lowe, B.S., M.D.
Robert B. Sasscer, A.B., M.D.
Henry D. Severn, M.D.
Joseph C. Sheehan, B.S., M.D.
Tracy N. Spencer, Jr., A.B., M.D.
John S. Stevens, A.B., M.D.

MERCY HOSPITAL DISPENSARY STAFF

Supervisor of Surgical Clinic	Raymond F. Helfrich
Assistant Surgeons	I. O. Ridgely H. F. Bongardt J. W. Nelson I. Ridgeway Trimble Simon H. Brager S. Demarco, Jr. E. Eugene Covington Richard T. Shackelford Meyer H. Zuravin ★Howard L. Zupnik Daniel R. Robinson Joseph V. Jerardi ★†Raymond J. Lipin †William M. Garlick
Supervisor of Genito-Urinary Clinic	Kenneth D. Legge
Assistant Genito-Urinary Surgeons	L. K. Fargo Francis W. Gillis
Orthopaedic Surgeons	Albertus Cotton Harry L. Rogers I. H. Maseritz Jason H. Gaskel
Supervisor of Medical Clinic	Maurice C. Pincoffs
Chiefs of Medical Clinic	Earl L. Chambers Sol Smith
Assistant Physicians	★Milton Stein Thomas G. Abbott Louis F. Klimes ★Irving Freeman
Chief of Cardiovascular Clinic	Thomas C. Wolff
Assistant Cardiologist	Harry Ashman
Chief of Metabolism Clinic	J. Sheldon Eastland
Assistant	Louis F. Klimes
Supervisor of Gastro-Enterology Clinic	§Julius Friedenwald
Gastro-Enterologists	Maurice Feldman Nathaniel Beck

Chief of Pediatric Clinic	Edgar Friedenwald
Pediatricians	G. Bowers Mansdorfer Jerome Fineman George Bayley
Supervisor of Neurological and Psychiatric Clinic	Andrew C. Gillis
Neurologists and Psychiatrists	★Harry Goldsmith Philip F. Lerner John W. Machen †George C. Medairy
Supervisor of Dermatology Clinic	Francis A. Ellis
Dermatologist	William D. Wolfe
Oncologist	James W. Nelson
Chief of Gynecology Clinic	Abram S. Samuels
Assistant Chief of Gynecology Clinic	Thomas K. Galvin
Gynecologists	Edward P. Smith Ernest S. Edlow H. L. Granoff John J. Erwin Frank K. Morris George A. Strauss, Jr.
Chief of Obstetrical Clinic	Edward P. Smith
Obstetricians	†William A. Dodd †Harry McBrine Beck ‡William B. Smith [3]‡Robert W. Stevens
Esophagoscopist	Waitman F. Zinn
Rhinologists and Laryngologists	Waitman F. Zinn Benjamin S. Rich Theodore A. Schwartz
Ophthalmologists and Otologists	M. Raskin F. A. Pacienza ★J. V. Jeppi
Supervisor of Proctology Clinic	Charles F. Blake
Proctologists	Eugene E. Covington Simon H. Brager
Dentists	J. D. Fusco B. Wallace Inman
Supervisor of Physiotherapy Clinic	Leon Hannan
Assistant Physiotherapist	Alice R. Hannan
Social Worker	Margaret Travers
Secretaries	Margaret O'Neill Eleanor Arnick

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[3] Military service.

MERCY HOSPITAL DISPENSARY REPORT

Year of 1940

Department	*New Cases*	*Old Cases*	*Total*
Cardiac	22	304	326
Dental	95	139	234
Diabetic	13	297	310
Eye and Ear	208	595	803
Gastro-Intestinal	61	426	487
Genito-Urinary	134	1,467	1,601
Gynecology	268	837	1,105
Medicine	609	2,171	2,780
Neurology	63	600	663
Nose and Throat	540	1,466	2,006
Orthopaedics	178	828	1,006
Pediatrics	200	622	822
Physiotherapy	91	1,030	1,121
Postnatal	288	75	363
Prenatal	356	2,004	2,360
Psychiatry	2	20	22
Rectal	40	73	113
Skin	205	600	805
Surgery	1,073	4,269	5,342
Tumor	3	3	6
Well Baby Clinic	63	45	108
Total	4,512	17,871	22,383

THE BALTIMORE CITY HOSPITALS

STAFF, 1941–1942

Parker J. McMillin, *Superintendent*

Consulting Chief Surgeon	Arthur M. Shipley, M.D., D.Sc.
Surgeon-in-Chief	Thomas B. Aycock, B.S., M.D.
Physician-in-Chief	John T. King, Jr., A.B., M.D.
Physician-in-Chief, Tuberculosis Hospital	H. Vernon Langeluttig, M.D.
Physician-in-Chief, Psychopathic Hospital	Esther L. Richards, M.D.
Obstetrician-in-Chief	Louis H. Douglass, M.D.
Pediatrician-in-Chief	T. Campbell Goodwin, A.B., M.D.
Pathologist-in-Chief	Frank B. Kindell, A.B., M.D.
Dental Surgeon-in-Chief	Lowell P. Henneberger, D.D.S.
Assistant Surgeon-in-Chief	Otto C. Brantigan, M.D.
Visiting Surgeons	James C. Owings, M.D. Harry C. Hull, M.D. Samuel McLanahan, Jr., A.B., M.D.
Consultant in Traumatic Surgery	Charles A. Reifschneider, M.D.
Consultant in General Surgery	I. Ridgeway Trimble, M.D.
Consultant in Peripheral Vascular Diseases	George H. Yeager, B.S., M.D.
Consultants in Plastic Surgery	Edward A. Kitlowski, M.D. Edward M. Hanrahan, Jr., M.D.
Assistant Visiting Surgeon	Luther E. Little, M.D.
Assistant Visiting Physician, Tuberculosis	†William H. Grenzer, M.D.

Assistant Visiting Psychiatrist	Thomas A. C. Rennie, M.D.
Visiting Obstetrician	J. Morris Reese, M.D.
Assistant Visiting Obstetricians	John M. Haws, B.S., M.D. Marius P. Johnson, A.B., M.D. John E. Savage, B.S., M.D. Hugh B. McNally, B.S., M.D. †William Dorman, M.D.
Visiting Pediatricians	William B. Stifler, M.D. B. Matthew Debuskey, M.D.
Assistant Visiting Pediatrician	William M. Seabold, A.B., M.D.
Visiting Gynecologist	J. Mason Hundley, Jr., M.A., M.D.
Assistant Visiting Gynecologists	‡Bernard C. Brack, M.D. Beverley Compton, M.D. John C. Dumler, B.S., M.D. John T. Hibbitts, M.D. Ernest I. Cornbrooks, Jr., M.D.
Assistant Chief Physician	C. Holmes Boyd, M.D.
Visiting Physicians	John W. Parsons, M.D. Louis A. M. Krause, M.D.
Visiting Cardiologist	Raymond Hussey, M.A., M.D.
Visiting Ophthalmologist	Frank B. Walsh, M.D.
Visiting Orthopaedic Surgeon	Allen F. Voshell, A.B., M.D.
Assistant Visiting Orthopaedic Surgeon	Henry F. Ullrich, M.D., D.Sc.
Visiting Laryngologists	Thomas R. O'Rourk, M.D. John Bordley, M.D.
Visiting Urologist	W. Houston Toulson, M.Sc., M.D.
Assistant Visiting Urologists	Hugh Jewett, M.D. [3]‡Harry S. Shelley, B.S., M.D. Howard B. Mays, M.D.
Visiting Neurological Surgeon	Charles Bagley, Jr., M.A., M.D.
Assistant Visiting Neurological Surgeon	Richard G. Coblentz, M.A., M.D.
Visiting Neurologists	Frank R. Ford, M.D. O. R. Langworthy, M.D.
Assistant Visiting Neurologists	Harry A. Teitelbaum, M.D. †Luis Ortega, M.D.
Visiting Proctologist	Monte Edwards, M.D.
Visiting Oncologists	Grant E. Ward, A.B., M.D. Murray M. Copeland, M.D.
Assistant Visiting Otologist	J. J. Belemer, M.D.
Visiting Dermatologist	Isaac R. Pels, M.D.
Visiting Dental Surgeon	H. Glenn Waring, D.D.S.
Assistant Visiting Dental Surgeons	L. W. Bimestefer, D.D.S. Joseph B. Berke, D.D.S. B. W. Miksinski, D.D.S.

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[3] Military service.

THE JAMES LAWRENCE KERNAN HOSPITAL AND INDUSTRIAL SCHOOL OF MARYLAND FOR CRIPPLED CHILDREN

STAFF, 1941–1942

Surgeon-in-Chief and Medical Director	Allen Fiske Voshell, A.B., M.D.
Attending Orthopaedic Surgeon and Roentgenologist	Albertus Cotton, A.M., M.D.
Associate Orthopaedic Surgeons	Moses Gellman, B.S., M.D. Harry Rogers, M.D. Winthrop M. Phelps, A.B., M.D. ‡Henry F. Ullrich, M.D., Sc.D.
Plastic Surgeon	Edward A. Kitlowski, A.B., M.D.
Aurist and Laryngologist	Benjamin S. Rich, A.B., M.D.
Dentist	M. E. Coberth, D.D.S.
Pediatrist	J. Edmund Bradley, B.S., M.D.
Consulting Surgeons	J. M. T. Finney, A.B., M.D., D.S.M., F.R.C.S. (Eng., Ire.) Hon. Arthur M. Shipley, M.D., Sc.D. C. Reid Edwards, A.B., M.D. Thomas B. Aycock, B.S., M.D.
Consulting Aurist and Laryngologist	Franklin B. Anderson, M.D. Edward A. Looper, M.D., D.Oph.
Consulting Neurological Surgeon	Charles Bagley, Jr., M.A., M.D.
Consulting Plastic Surgeon	John Staige Davis, B.Sc., M.D.
Consulting Physicians	Thomas R. Brown, A.B., M.D. Lewellys F. Barker, A.B., M.D.
Consulting Dermatologists	Harry M. Robinson, M.D. Leon Ginsberg, M.D.
Consulting Neurologists	Irving J. Spear, M.D. R. V. Seliger, M.D.
Consulting Pediatrist	Benjamin Tappan, A.B., M.D.
Consulting Dentist	Harry B. McCarthy, D.D.S.
Consulting Opthalmologists	Harry Friedenwald, A.B., M.D., D.H.L., D.Sc. William Tarun, M.D.
Consulting Pathologist	Hugh R. Spencer, M.D.
Consulting Roentgenologists	Howard E. Ashbury, A.B., M.D. Henry Walton, M.D.
Resident Orthopaedic Surgeons	‡H. K. Magill, M.D. †R. C. Bell, M.D.
Superintendent	Miss Maud M. Gardner, R.N.
Dispensary and Social Service Nurse	Mrs. Evelyn Byrd Zatf, R.N.
Physical Therapists and X-ray Technician	Miss Jane Ewing, P.T., Reg. Miss Jane Anderson, R.N. Mrs. Georgiana Wisong
Occupational Therapist	Miss Muriel Zimmerman, O.T.
Instructor in Grammar School	Mrs. Laura Siemon

HISTORY OF THE SCHOOL OF MEDICINE

The present School of Medicine, with the title University of Maryland School of Medicine and College of Physicians and Surgeons, is the result of a consolidation and merger of the University of Maryland School of Medicine with the Baltimore Medical College (1913) and the College of Physicians and Surgeons of Baltimore (1915).

The School of Medicine of the University of Maryland is one of the oldest foundations for medical education in America, ranking fifth in point of age among the medical colleges of the United States. It was organized in 1807, and chartered in 1808, under the name of the College of Medicine of Maryland, and its first class was graduated in 1810. In 1812 the College was empowered by the Legislature to annex three other colleges or faculties: Divinity, Law, and Arts and Sciences; and the four colleges thus united were "constituted an University by the name and under the title of the University of Maryland."

The original building of the Medical School at the N. E. corner of Lombard and Greene Streets, was erected in 1812. It is the oldest structure in this country from which the degree of doctor of medicine has been granted annually since its erection. In this building were founded one of the first medical libraries and one of the first medical school libraries in the United States.

At this Medical School for the first time in America, dissection was made a compulsory part of the curriculum, and independent chairs for the teaching of gynecology and pediatrics (1867), and of ophthalmology and otology (1873), were installed.

This School of Medicine was one of the first to provide for adequate clinical instruction by the erection of its own hospital in 1823. In this hospital intramural residency for senior students was established for the first time.

In 1913, a merger was brought about with the Baltimore Medical College, an institution of thirty-two years' growth. By this association the facilities of the School of Medicine were enlarged in faculty, equipment and hospital connection.

The College of Physicians and Surgeons was incorporated in 1872, and established on Hanover Street in a building afterward known as the *Maternité*, the first obstetrical hospital in Maryland. In 1878 union was effected with the Washington University School of Medicine, in existence since 1827, and the college was removed to Calvert and Saratoga Streets. Through the consolidation with the College of Physicians and Surgeons, in 1915, medical control of the teaching beds in the City Hospital, now the Mercy Hospital, was obtained.

BUILDINGS AND FACILITIES

The original medical building at the N. E. corner of Lombard and Greene Streets houses the office of the Dean, the Conference Room of the Faculty, and two lecture halls.

The Administration Building, to the east of the original building, contains the Baltimore offices of the Registrar and the Director of Admissions and two lecture halls.

The laboratory building at 31 South Greene Street is occupied by the departments of Pathology, Bacteriology and Biochemistry.

The Frank C. Bressler Research Laboratory provides the departments of Anatomy, Histology and Embryology, Pharmacology, Physiology and Clinical Pathology with facilities for teaching and research. It also houses the research laboratories of the clinical departments, animal quarters, a laboratory for teaching Operative Surgery, a lecture hall and the Bressler Memorial Room.

This building was erected in 1939–1940 at 29 South Greene Street opposite the University Hospital. It was built with funds left to the School of Medicine by the late Frank C. Bressler, an alumnus, supplemented by a grant from the Federal government. The structure, in the shape of an I, extends east from Greene Street, just north of the original building.

MEDICAL LIBRARY

Carl W. F. Hintz, A.B.	University Librarian
Ruth Lee Briscoe	Medical Librarian
Julia E. Wilson, B.S.	Assistant Medical Librarian

The Medical Library of the University of Maryland, founded in 1813 by the purchase of the collection of Dr. John Crawford, now numbers 21,500 volumes and several thousand pamphlets and reprints. Over three hundred of the leading medical journals, both foreign and domestic, are received regularly. The library is housed in Davidge Hall, a comfortable and commodious building in close proximity to classrooms and laboratories, and is open daily for the use of members of the faculty, the student body and the profession generally. Libraries pertaining to particular phases of medicine are maintained by several departments of the medical school.

The library of the Medical and Chirurgical Faculty of Maryland and the Welch Medical Library are open to students of the medical school without charge. Other libraries of Baltimore are the Peabody Library and the Enoch Pratt Free Library.

DISPENSARY BUILDING

The old hospital building has been remodeled and is occupied by the Out-patient Department. Thus the students have been provided with a splendidly appointed group of clinics for their training in out-patient work. All departments of clinical training are represented in this remodeled building and all changes have been predicated on the teaching function for which this department is intended.

The office of the Medical School Physician is located in this building.

The Department of Art also occupies quarters here.

UNIVERSITY HOSPITAL

The University Hospital, which is the property of the University of Maryland, is the oldest institution for the care of the sick in the state of Maryland. It was opened in September, 1823, under the name of the Baltimore Infirmary, and at that time consisted of but four wards, one of which was reserved for patients with diseases of the eye.

In 1933–1934 the new University Hospital was erected, and patients were admitted to this building in November 1934. The new hospital is situated at the southwest corner of Redwood and Greene Streets, and is consequently opposite the medical school buildings. The students, therefore, are in close proximity and little time is lost in passing from the lecture halls and laboratories to the clinical facilities of the new building.

This new building, with its modern planning, makes a particularly attractive teaching hospital and is a very valuable addition to the clinical facilities of the medical school.

The new hospital has a capacity of 435 beds and 50 bassinets devoted to general medicine, surgery, obstetrics, pediatrics, and the various medical and surgical specialties.

The teaching zone extends from the second to the eighth floor and comprises wards for surgery, medicine, obstetrics, pediatrics, and a large clinical lecture hall. There are approximately 270 beds available for teaching.

The space of the whole north wing of the second floor is occupied by the Department of Roentgenology. The east wing houses clinical pathology and special laboratories for clinical microscopy, biochemistry, bacteriology, and an especially well appointed laboratory for students' training. The south wing provides space for electro-cardiographic and basal metabolism departments, with new and very attractive air-conditioned or oxygen therapy cubicles. The west wing contains the Departments of Rhinolaryngology and Bronchoscopy, Industrial Surgery, Ophthalmology, and Male and Female Cystoscopy.

The third and fourth floors each provide two medical and two surgical wards. The fifth floor contains two wards for pediatrics, and on the sixth floor there are two wards for obstetrics. Each ward occupies the space of one wing of the hospital.

On the seventh floor is the general operating suite, the delivery suite, and the central supply station. The eighth floor is essentially a students' floor and affords a mezzanine over the operating and delivery suites, and a students' entrance to the clinical lecture hall.

In the basement there is a very well appointed Pathological Department with a large teaching autopsy room and its adjunct service of instruction of students in pathological anatomy.

The hospital receives a large number of accident patients because of its proximity to the largest manufacturing and shipping districts of the city.

The obstetrical service is particularly well arranged and provides accommodation for forty ward patients. This service, combined with an extensive home service, assures the student abundant obstetrical training.

During the year ending December 31, 1940, 1429 cases were delivered in the hospital and 968 cases in the outdoor department. Students in the graduating class observed at least thirty-five cases, each student being required to deliver at least ten patients in their homes.

The dispensaries associated with the University Hospital and the Mercy Hospital are organized upon a uniform plan in order that the teaching may be the same in each. Each dispensary has the following departments: Medicine, Surgery, Pediatrics, Ophthalmology, Otology, Genito-Urinary, Gynecology, Gastro-

Enterology, Neurology, Orthopaedics, Proctology, Dermatology, Laryngology, Rhinology, Cardiology, Tuberculosis, Psychiatry, Oral Surgery and Oncology.

All students in their junior year work each day during one-third of the year in the Departments of Medicine and Surgery of the dispensaries. In their senior year, all students work one hour each day in the special departments.

MERCY HOSPITAL

The Sisters of Mercy first assumed charge of the Hospital at the corner of Calvert and Saratoga Streets, then owned by the Washington University, in 1874. By the merger of 1878 the Hospital came under the control of the College of Physicians and Surgeons, but the Sisters continued their work of ministering to the patients.

In a very few years it became apparent that the City Hospital, as it was then called, was much too small to accommodate the rapidly growing demands upon it. However, it was not until 1888 that the Sisters of Mercy, with the assistance of the Faculty of the College of Physicians and Surgeons, were able to lay the cornerstone of the present hospital. This building was completed and occupied late in 1889. Since then the growing demands for more space have compelled the erection of additions, until now there are accommodations for 310 patients.

In 1909 the name was changed from The Baltimore City Hospital to Mercy Hospital.

The clinical material in the free wards is under the exclusive control of the Faculty of the University of Maryland School of Medicine and College of Physicians and Surgeons.

THE BALTIMORE CITY HOSPITALS

The clinical facilities of the School of Medicine have been largely increased by the liberal decision of the Department of Public Welfare to allow the use of the wards of these hospitals for medical education. The autopsy material also is available for student instruction.

Members of the junior class make daily visits to these hospitals for clinical instruction in medicine, surgery, and the specialties.

The Baltimore City Hospitals consist of the following separate divisions:

The General Hospital, 400 beds, 60 bassinets.
The Hospital for Chronic Cases, 575 beds.
The Hospital for Tuberculosis, 280 beds.
The Psychopathic Hospital, 85 beds.
Infirmary (Home for Aged) 1053 beds.

THE JAMES LAWRENCE KERNAN HOSPITAL AND INDUSTRIAL SCHOOL OF MARYLAND FOR CRIPPLED CHILDREN

This institution is situated on an estate of 75 acres at Dickeyville. The site is within the northwestern city limits and of easy access to the city proper.

The location is ideal for the treatment of children, in that it affords all the advantages of sunshine and country air.

A hospital unit, complete in every respect, offers all modern facilities for the care of any orthopaedic condition in children.

The hospital is equipped with 100 beds—endowed, and city and state supported.

The Orthopaedic Dispensary at the University Hospital is maintained in closest affiliation and cares for the cases discharged from the Kernan Hospital. The Physical Therapy Department is very well equipped with modern apparatus and trained personnel. Occupational therapy has been fully established and developed under trained technicians.

THE BALTIMORE EYE, EAR, AND THROAT HOSPITAL

This institution was first organized and operated in 1882 as an outgrowth of the Baltimore Eye and Ear Dispensary, which closed on June 14, 1882. The name then given to the new hospital was The Baltimore Eye and Ear Charity Hospital. It was located at the address now known as 625 W. Franklin St. The out-patient department was opened on September 18th, 1882 and the hospital proper on November 1st of the same year. In 1898 a new building afforded 24 free beds and 8 private rooms; by 1907 the beds numbered 47; at present there are 60 beds, 29 of which are free. In 1922 the present hospital building at 1214 Eutaw Place was secured and in 1926 the dispensary was opened. In 1928 a clinical laboratory was installed. During 1940 the out-patient visits numbered 23,498.

Through the kindness of the Hospital Board and Staff, our junior students have access to the dispensary which they visit in small groups for instruction in ophthalmology.

REQUIREMENTS FOR ADMISSION

METHOD OF MAKING APPLICATION

Application forms may be filed beginning October 1 for the following September classes. These forms may be secured from the Committee on Admissions, School of Medicine, University of Maryland, Baltimore.

APPLICATION FOR ADMISSION TO THE FIRST YEAR

Application for admission is made by filing the required form and by having all pertinent data sent directly to the Committee on Admissions, in accordance with the instructions accompanying the application.

APPLICATION FOR ADMISSION TO ADVANCED STANDING

Students who have attended approved medical schools are eligible to file applications for admission to the second- and third-year classes. These applicants must be prepared to meet the current first-year entrance requirements in addition to presenting acceptable medical school credentials, and a medical school record based on courses which are quantitatively and qualitatively equivalent to similar courses in this school.

Application to advanced standing is made in accordance with the instructions accompanying the application form.

MINIMUM REQUIREMENTS FOR ADMISSION

The minimum requirements for admission to the School of Medicine are:

(a) Graduation from an approved secondary school, or the equivalent in entrance examinations, and

(b) Three calendar years of acceptable premedical credit earned in an approved college of arts and sciences. The quantity and quality of this preprofessional course of study shall be not less than that required for recommendation by the institution where the premedical courses are being, or have been, studied.

The premedical curriculum shall include basic courses in

English
Biology
Inorganic Chemistry
Organic Chemistry
Physics
French or German

and such elective courses as will complete a balanced three-year schedule of study.

The elective courses should be selected from the following three groups:

Humanities	*Natural Sciences*	*Social Sciences*
English (an advanced course in English composition should be taken, if possible)	Comparative Vertebrate Anatomy	Economics
Scientific German or French (A reading knowledge of either language is desirable, although German is preferred)	Embryology	History
Philosophy	Quantitative Analysis	Political Science
	Physical Chemistry	Psychology (a basic course is desirable)
	Mathematics	Sociology, etc.

Careful attention should be given to the selection of elective courses in the natural sciences. Accordingly, it is suggested that the elective list given above be a guide in this connection and that the remainder of the college credits be accumulated from courses designed to promote a broad cultural development. Students should avoid the inclusion of college courses in subjects that occur in the medical curriculum, for example, histology, histological technique, human anatomy, bacteriology, physiology, neurology, physiological chemistry, etc.

It is not intended that these suggestions be interpreted to restrict the education of students who exhibit an aptitude for the natural sciences or to limit the development of students who plan to follow research work in the field of medicine.

In accepting candidates for admission, preference will be given to those applicants who have acceptable scholastic records in secondary school and college, satisfactory scores in the Medical Aptitude Test, favorable letters of recommendation from their premedical committees, or from one instructor in each of the departments of biology, chemistry, and physics, and who in all other respects give every promise of becoming successful students and physicians of high standing.

Those candidates for admission who are accepted will receive certificates of entrance from the Director of Admissions of the University.

COMBINED COURSE IN ARTS AND SCIENCES, AND MEDICINE

A combined seven years' curriculum leading to the degrees of Bachelor of Science and Doctor of Medicine is offered by the University of Maryland. The first three years are taken in residence in the College of Arts and Sciences at College Park, and the last four years in the School of Medicine in Baltimore. (See University catalogue for details of quantitative and qualitative premedical course requirements.)

Upon the successful completion of the first year in the School of Medicine, and upon the recommendation of the Dean, the degree of Bachelor of Science may be conferred by the College of Arts and Sciences at the commencement following the *second year* of residence.

STATE MEDICAL STUDENT QUALIFYING CERTIFICATES

Candidates for admission who live in or expect to practice medicine in Pennsylvania, New Jersey or New York, should apply to their respective state boards of education for medical student qualifying certificates (Pennsylvania and New Jersey) or approval of applications for medical student qualifying certificates (New York).

Those students who are accepted must file satisfactory State certificates in the office of the Committee on Admissions, School of Medicine, before registration. No exceptions will be made to this requirement.

Addresses of the State Certifying Offices

Director of Credentials Section, Pennsylvania Department of Public Instruction, Harrisburg, Pa.

Chief of the Bureau of Credentials, New Jersey Department of Public Instruction, Trenton, N. J.

Supervisor of Qualifying Certificates, The State Education Department, Examinations and Inspections Division, Albany, N. Y.

DEFINITION OF RESIDENCE STATUS OF STUDENTS*

Students who are minors are considered to be resident students if, at the time of their registration, the parents* have been residents of this State for at least one year.

Adult students are considered to be resident students if, at the time of their registration, they have been residents of this State for at least one year, provided such residence has not been acquired while attending any school or college in Maryland.

The status of the residence of a student is determined at the time of this first registration in the university and may not thereafter be changed by him unless, in the case of a minor, his parents* move to and become legal residents of this state by maintaining such residence for at least one full calendar year. However, the right of the student (minor) to change from a non-resident to a resident status must be established by him prior to registration for a semester in any academic year.

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* The term "parents" includes persons who, by reason of death or other unusual circumstances, have been legally constituted the guardians of or stand in loco parentis to such minor students.

FEES

Matriculation fee (paid once)	$10.00
Tuition fee (each year)—Residents of Maryland	450.00
Tuition fee (each year)—Non-Residents	600.00
Laboratory fee (each year)	25.00
Conditioned examination fee (each subject)	5.00
Student health service fee (each year)	10.00
Student activities fee (each year)	5.00
Maintenance and service fee (each year)	5.00
Graduation fee	15.00
Transcript fee to graduates. First copy gratis, thereafter, each copy	1.00

RULES FOR PAYMENT OF FEES

No fees are returnable.

The above fees apply to all students who matriculate in the School of Medicine in any class for the session beginning September 25, 1941.

Make all checks or money orders payable to the University of Maryland.

When offering checks or money orders in payment of tuition and other fees, students are requested to have them drawn in the exact amount of such fees. Personal checks whose face value is in excess of the fees due will be accepted for collection only.

Acceptance.—The matriculation fee of $10.00 is payable on acceptance. In addition, a deposit on tuition of $50.00 is required of accepted applicants before the expiration date specified in the offer of acceptance. This $60.00 deposit is not returnable and will be forfeited if the applicant fails to register, or it will be applied to the applicant's first semester's charges on registration.

Registration.—All students, after proper certification, are required to register at the Office of the Registrar. (See calendar in front of this bulletin for dates for the payments of fees, and the note regarding late registration fee.)

One-half of the tuition fee, the laboratory fee, the student health fee, the student activities fee, and the maintenance and service fee are payable on the date specified for registration for the first semester.

The remainder of the tuition fee shall be paid on the date of registration for the second semester. Fourth year students shall pay the graduation fee, in addition, at this registration.

PENALTY FOR NON-PAYMENT OF FEES

If semester fees are not paid in full on the specified registration dates, a penalty of $5.00 will be added. This penalty also applies to deferred payments.

If a satisfactory settlement, or an agreement for settlement, is not made with the Dean within ten days after a payment is due, the student automatically is debarred from attendance on classes and will forfeit the other privileges of the School of Medicine.

CONDITIONED EXAMINATION FEE

A student who is eligible to conditioned examinations must notify the Dean two weeks in advance of his intention to take the examinations. He will pay the comptroller $5.00 for each subject in which he is to be examined, and he must present the receipt to the faculty member giving the examination before he will be permitted to take the examination.

MAINTENANCE AND SERVICE FEE

This fee pays for the maintenance of loan collections, provides photographs for departmental requests and state board applications, pays room-rent for the third- and fourth-year students when on outside obstetrics, and furnishes student invitations to the Pre-Commencement Exercises.

PERSONAL EXPENSES

The following estimates of personal expenses for the academic year of eight months have been prepared by students, and are based upon actual experience. In addition to these the student must bear in mind the expenditure for a microscope.

	Low	*Average*	*Liberal*
Books	$50	$75	$100
College incidentals	20	20	20
Board, eight months	200	250	275
Room rent	64	80	100
Clothing and laundry	50	80	150
All other expenses	25	50	75
Total	$409	$556	$720

STUDENT HEALTH SERVICE

T. Nelson Carey, M.D. .... Director, Student Health Service

The Medical Council has made provision for the systematic care of students in the Medical School, according to the following plan:

1. *Preliminary Examination*—All new students will be examined during the first week of the semester. Notice of the date, time, and place of the examination will be announced to the classes and on the bulletin board. The passing of this physical examination is necessary before final acceptance of any student.

2. *Medical Attention*—Students in need of medical attention will be seen by the school physician, Dr. T. N. Carey, in his office at the medical school, between 4 and 5 P.M., daily, except Saturday and Sunday. In case of necessity, students will be seen at their homes.

3. *Hospitalization*—If it becomes necessary for any student to enter the hospital during the school year, the Medical Council has arranged for the payment of part or all of his hospital expenses, depending on the length of his stay and special expenses incurred. This applies only to students admitted through the school physician's office.

4. *Physical Defects*—Prospective students are advised to have any known physical defects corrected before entering school in order to prevent loss of time which later correction might incur.

Each new matriculate is required to undergo an eye examination at the hands of an oculist (Doctor of Medicine) before entering the School of Medicine. In many students long study hours bring out unsuspected eye defects which cause much loss of time and inefficiency in study if not corrected until after school work is under way.

An eye examination will be required beginning September, 1941.

5. *Limitations*—It is not the function of this service to treat chronic conditions contracted by students before admission, nor to extend treatment to acute conditions arising in the period between academic years, unless the school physician recommends this service.

GENERAL RULES

All rules, as well as fees, relate to the year ending June 6, 1942 only. The right is reserved to make changes in the curriculum, the requirements for graduation, the fees and in any of the regulations whenever the university authorities deem it expedient.

ADVANCEMENT AND GRADUATION

1. All students are required to take the spring examinations unless excused by the Dean.
2. No student will be permitted to pass to a higher class with conditions.
3. Should a student be required to repeat any year in the course, he must pay regular fees.
4. A student failing in final examinations for graduation at the end of the fourth year will be required to repeat the entire course of the fourth year and to take examinations in such other branches as may be required, should he again be permitted to enter the school as a candidate for graduation.
5. The general fitness of a candidate for graduation, as well as the results of his examinations, will be taken into consideration by the faculty.

EQUIPMENT

6. At the beginning of the first year, all students must be prepared to purchase microscopes of a satisfactory type equipped with a mechanical stage and a substage lamp. The cost is about $177.00, when new.

A standard microscope of either Bausch & Lomb, Leitz, Spencer, or Zeiss make, fitted with the following attachments, will fill the requirements:

Oculars: 10 x and 5 x.
Triple nose piece with 16 mm., 4 mm., and 1.9 mm. 125 N.A. oil immersion lens.
Wide aperture stage with quick screw condenser.

All used microscopes are subject to inspection and approval before their use in the laboratory is permitted. The student is cautioned against the purchase of such an instrument before its official approval by the school.

7. Students in the third- and fourth-year classes are required to furnish their own ophthalmoscopes.
8. Third- and fourth-year students are required to provide haemocytometers.

STATE QUALIFYING CERTIFICATES

9. Candidates for admission who live in or expect to practice medicine in Pennsylvania, New Jersey or New York must file State certificates in the office of the Committee on Admissions, School of Medicine, before registration. No exception will be made to this rule.

EYE EXAMINATION BEFORE ADMISSION

(*Effective September, 1941*)

10. Each new matriculate in each class is required to present to the Committee on Admissions a certificate from an oculist, (a graduate in medicine) that the matriculate's eyes have been examined and are in condition, with or without glasses as the case may be, to endure the strain of close and intensive reading.

It is required that this examination be completed within three months prior to registration and that the certificate be mailed to the Committee on Admissions not later than one month before registration.

AWARDING OF COMBINED DEGREES

11. Students entering the School of Medicine on a three-year requirement basis from colleges which usually grant a degree on the successful completion of the first year of medicine, are restricted by the following rule:

> Upon successful completion of the first year in the School of Medicine, and upon recommendation of the Dean of the School of Medicine, the degree of Bachelor of Science may be conferred by a College of Arts and Sciences following the student's *second year* of residence in the School of Medicine.

COST OF TRANSCRIPTS

12. Graduates will receive the first transcript of record without charge. Subsequent copies will cost one dollar each.

PRIZES

THE FACULTY PRIZE

Each year the faculty awards a gold medal to the graduate who during the four years of his course has shown the greatest proficiency in preparing for the practice of medicine. The five candidates standing next in order will be awarded certificates of honor.

THE DR. A. BRADLEY GAITHER MEMORIAL PRIZE

A prize of $25.00 is given each year by Mrs. A. Bradley Gaither as a memorial to the late Dr. A. Bradley Gaither, to the student in the senior class doing the best work in genito-urinary surgery.

SCHOLARSHIPS

All scholarships are assigned for one academic year, unless specifically reawarded on consideration of an application.

Official application forms are obtainable at the Dean's office, where they should be filed before July 1, for the ensuing academic year.

THE DR. SAMUEL LEON FRANK SCHOLARSHIP

(Value $100.00)

This scholarship was established by Mrs. Bertha Rayner Frank as a memorial to the late Dr. Samuel Leon Frank, an alumnus of this university.

It is awarded by the Trustees of the Endowment Fund of the University each year upon nomination by the Medical Council "to a medical student of the University of Maryland, who in the judgment of said Council, is of good character and in need of pecuniary assistance to continue his medical course."

This scholarship is awarded to a second, third or fourth year student who has successfully completed one year's work in this school. No student may hold this scholarship for more than two years.

THE CHARLES M. HITCHCOCK SCHOLARSHIPS

(Value $100.00 each)

Two scholarships were established from a bequest to the School of Medicine by the late Charles M. Hitchcock, M.D., an alumnus of the university.

These scholarships are awarded annually by the Trustees of the Endowment Fund of the University, upon nomination by the Medical Council, to students who have meritoriously completed the work of at least the first year of the course in medicine, and who present to the council satisfactory evidence of a good moral character and of inability to continue the course without pecuniary assistance.

THE RANDOLPH WINSLOW SCHOLARSHIP

(Value $100.00)

This scholarship was established by the late Randolph Winslow, M.D., LL.D.

It is awarded annually by the Trustees of the Endowment Fund of the University, upon nomination by the Medical Council, to a "needy student of the Senior, Junior, or Sophomore Class of the Medical School."

"He must have maintained an average grade of 85% in all his work up to the time of awarding the scholarship."

"He must be a person of good character and must satisfy the Medical Council that he is worthy of and in need of assistance."

THE DR. LEO KARLINSKY MEMORIAL SCHOLARSHIP

(Value $125.00)

This scholarship was established by Mrs. Ray Mintz Karlinsky as a memorial to her husband, the late Dr. Leo Karlinsky, an alumnus of the university.

It is awarded annually by the Trustees of the Endowment Fund of the University, upon the nomination of the Medical Council, to "a needy student of the Senior, Junior or Sophomore Class of the Medical School."

He must have maintained in all his work up to the time of awarding the scholarship a satisfactory grade of scholarship.

He must be a person of good character and must satisfy the Medical Council that he is worthy of and in need of assistance.

THE UNIVERSITY SCHOLARSHIP

A scholarship which entitles the holder to exemption from payment of tuition fee for the year, is awarded annually by the Medical Council to a student of the senior class in need of assistance who presents to the Medical Council satisfactory evidence of good character and scholarship.

THE FREDERICA GEHRMANN SCHOLARSHIP

(Value $200.00)

(Not open to holders of Warfield and Cohen Scholarships)

This scholarship was established by the bequest of the late Mrs. Frederica Gehrmann and is awarded to a third-year student who at the end of the second year has passed the best practical examinations in physiology, pharmacology, pathology, bacteriology, immunology, serology, surgical anatomy and neuroanatomy.

THE CLARENCE AND GENEVRA WARFIELD SCHOLARSHIPS

(Value $300.00 each)

There are five scholarships established by the regents from the income of the fund bequeathed by the will of Dr. Clarence Warfield.

Terms and Conditions: These scholarships are available to students of any of the classes of the course in medicine. Preference is given to students from the counties of the state of Maryland which the Medical Council may from time to time determine to be most in need of medical practitioners.

Any student receiving one of these scholarships must agree, after graduation and a year's internship, to undertake the practice of medicine, for a term of two years, in the county to which the student is accredited, or in a county selected by the council. In the event that a student is not able to comply with the condition requiring him to practice in the county to which he is accredited by the council, the money advanced by the regents shall be refunded by the student.

THE ISRAEL AND CECELIA E. COHEN SCHOLARSHIP

(Value $150.00)

This scholarship was established by the late Eleanor S. Cohen in memory of her parents, Israel and Cecelia E. Cohen. Terms and conditions: This scholarship will be available to students of any one of the classes of the course in medicine; preference is given to students of the counties in the state of Maryland which the Medical Council may from time to time determine to be most in need of medical practitioners. Any student receiving one of these scholarships must, after graduation and a year's internship, agree to undertake the practice of medicine for a term of two years in the county to which the student is accredited, or in a county selected by the council. In the event that a student is not able to comply with the condition requiring him to practice in the county to which he is accredited by the council, the money advanced by the regents shall be refunded.

THE DR. HORACE BRUCE HETRICK SCHOLARSHIP

(Value $125.00)

This scholarship was established by Dr. Horace Bruce Hetrick as a memorial to his sons, Bruce Hayward Hetrick and Augustus Christian Hetrick. It is to be awarded by the Medical Council to a student of the senior class.

THE MEDICAL ALUMNI ASSOCIATION SCHOLARSHIP

(Value $300.00)

This scholarship was established by The Medical Alumni Association of the University of Maryland.

It is awarded, on recommendations of the Board of Directors of the Medical Alumni Association and of the Chairman of the Committee on Admissions of the School of Medicine, to a student of the first year who must furnish satisfactory evidence of good character and scholastic accomplishment and of his inability to pursue the medical course without pecuniary support.

ORGANIZATION OF THE CURRICULUM

The following curriculum is the result of a thorough revision of teaching in this school in order to meet modern requirements. The multiplication of specialties in medicine and surgery necessitates a very crowded course and the introduction of electives will very soon be depended on to solve some of the difficulties.

The curriculum is organized under eleven departments.

1. Anatomy (including Histology and Embryology).
2. Physiology.
3. Bacteriology and Immunology.
4. Biological Chemistry.
5. Pharmacology and Materia Medica.
6. Pathology.
7. Medicine (including Medical Specialties).
8. Surgery (including Surgical Specialties).
9. Obstetrics.
10. Gynecology.
11. Ophthalmology.

The instruction is given in four years of graded work.

Several courses of study extend through two years or more, but in no case are the students of different years thrown together in the same course of teaching.

The first and second years are devoted largely to the study of the structures and functions of the normal body. Laboratory work occupies most of the student's time during these two years.

Some introductory instruction in medicine and surgery is given in the second year. The third and fourth years are almost entirely clinical.

A special feature of instruction in the school is the attempt to bring together teacher and student in close personal relationship. In many courses of instruction the classes are divided into small groups and a large number of instructors insures attention to the requirements of each student.

In most courses the final examination as the sole test of proficiency has disappeared and the student's final grade is determined largely by partial examinations, recitations and assigned work carried on throughout the course.

DEPARTMENT OF GROSS ANATOMY

Eduard Uhlenhuth	Professor of Anatomy
Frank H. J. Figge	Associate Professor of Gross Anatomy
James C. Plagge	Assistant Professor of Gross Anatomy
E. Eugene Covington	Associate in Gross Anatomy
Otto C. Brantigan	Associate in Gross Anatomy
Harry C. Hull	Associate in Surgery
Thurston R. Adams	Instructor in Surgery
Harry A. Teitelbaum	Instructor in Gross Anatomy
Herbert E. Reifschneider	Assistant in Surgery
George H. Brouillet	Assistant in Surgery
Harry C. Bowie	Assistant in Surgery and Gross Anatomy

Gross Anatomy. *First Year.* Four lectures and conferences and nineteen hours of laboratory (dissection of the human subject) are given each week during the first semester. Drs. Uhlenhuth, Figge, Plagge, Covington, Teitelbaum and Bowie.

Total hours: 350.

Topographic and Surgical Anatomy. *Second Year.* The course is designed to bridge the gap between anatomy in the abstract and clinical anatomy as applied to the study and practice of medicine and surgery. Students are required to demonstrate all points, outlines, and regions on the cadaver. Underlying regions are dissected to bring out outlines and relations of structures.

Two lectures and two laboratory periods are given each week during the second semester. Drs. Brantigan, Hull, Adams, Brouillet, H. E. Reifschneider and Bowie.

Total hours: 96.

Advanced Anatomy (*elective course*). Selected problems in gross anatomy. This course is intended primarily to offer to the sophomore student an opportunity of extending the knowledge secured in the freshman course. Drs. Uhlenhuth, Figge, Plagge and Brantigan.

Total hours: 64.

In addition to the above courses, facilities for special anatomical problems are offered to the more advanced student and physician.

Graduate Courses. Consult the catalogue of the Graduate School for descriptions of the graduate courses offered by members of the staff.

DEPARTMENT OF HISTOLOGY AND EMBRYOLOGY

Carl L. Davis	Professor of Anatomy
O. G. Harne	Associate Professor of Histology
John F. Lutz	Assistant Professor of Histology
	Instructor in Histology
	Instructor in Histology

First Year. The course in histology is divided equally between the study of the fundamental tissues and that of organs. Throughout the entire course the embryology of the part being studied precedes the study of the fully developed tissue. Thus embryology becomes a correlated part of the whole subject of microscopic anatomy and not an independent subject.

Each student is furnished a set of histological slides, previously prepared in our own laboratory, thus insuring a uniform and satisfactory quality of material for study and permitting the time of the student to be expended in the study of material rather than in the technic of its preparation. The object of the course is to present the evidence of function as shown by the structure of tissues and organs. Drs. Davis, Lutz and Harne.

An optional laboratory course is offered. This supplements the required course giving laboratory experience which can not be incorporated in the former. No added charge is made for the course.

Total hours: 150.

NEURO-ANATOMY

First Year. Neuro-anatomy embraces a study of the fundamental structure of the central nervous system as applied to its function. An abundance of material permits of individual dissection of the human brain. A series of appropriately stained sections of the human brain stem is furnished each student for the microscopic study of the internal structure of the nervous system. Drs. Davis, Lutz and Harne.

Total hours: 100.

Graduate Courses. Consult the catalogue of the Graduate School for descriptions of the graduate courses offered by members of the staff.

DEPARTMENT OF PHYSIOLOGY

William R. Amberson	Professor of Physiology
Dietrich Conrad Smith	Associate Professor of Physiology
Robert H. Oster	Assistant Professor of Physiology
James E. P. Toman	Instructor in Physiology
Joye E. Jacobs	Assistant in Physiology
Guy M. Everett	Weaver Fellow in Physiology

Five lectures, one conference and two laboratory periods a week, September to January inclusive. The fundamental concepts of physiology are presented with special reference to mammalian problems.

Total hours: 224.

Graduate Courses. Consult the catalogue of the Graduate School for descriptions of the graduate courses offered by members of the staff.

DEPARTMENT OF BACTERIOLOGY AND IMMUNOLOGY

Frank W. Hachtel	Professor of Bacteriology
James G. McAlpine	Associate Professor of Bacteriology
★Henry F. Buettner	Instructor in Bacteriology
H. Edmund Levin	Instructor in Bacteriology
Milton S. Sacks	Assistant in Bacteriology

The principles of general bacteriology are taught by quiz, conference, and lecture.

Instruction in bacteriology is given in the laboratory to students of the second year class during the first semester. This includes the methods of preparation of culture media, the study of pathogenic bacteria, and the bacteriological examination of water and milk. The bacteriological diagnosis of communicable diseases is also included.

The principles of immunology are presented by means of quizzes, conferences and lectures to the second year class in the second semester.

The course includes a consideration of infection and immunity, the nature and action of the various antibodies, complement fixation and flocculation tests, hypersensitiveness, and the preparation of bacterial vaccines.

Experiments are carried out by the class in the laboratory. During the latter half of the semester the class is divided into sections.

Total hours: Bacteriology 120.
Immunology 72.

Graduate Courses. Consult the catalogue of the Graduate School for descriptions of the graduate courses offered by members of the staff.

DEPARTMENT OF BIOLOGICAL CHEMISTRY

H. Boyd Wylie	Professor of Biological Chemistry
Emil G. Schmidt	Associate Professor of Biological Chemistry
Frank N. Ogden	Associate in Biological Chemistry
Ann Virginia Brown	Assistant in Biological Chemistry

This course is designed to present the principles of biological chemistry and to indicate their applications to the clinical aspects of medicine. The phenomena of living matter and its chief ingredients, secretions and excretions are discussed in lectures and conferences and examined experimentally. Training is given in biochemical methods of investigation. Total hours: 208.

Graduate Courses. Consult the catalogue of the Graduate School for descriptions of the graduate courses offered by members of the staff.

DEPARTMENT OF PHARMACOLOGY

John C. Krantz, Jr.	Professor of Pharmacology
C. Jelleff Carr	Associate Professor of Pharmacology
William Ellsworth Evans, Jr.	Assistant Professor of Pharmacology
Ruth Musser	Instructor in Pharmacology
Marius P. Johnson	Assistant in Pharmacology
Henry Wollenweber	Assistant in Pharmacology
William G. Harne	Demonstrator in Pharmacology
Frederick K. Bell	U. S. Pharmacopoeia Fellow
Sylvan Forman	Fellow in Pharmacology
Fred W. Ellis	Fellow in Pharmacology
Wilson C. Grant	Research Assistant in Pharmacology, International Cancer Research Foundation
Maurice Rath	Fellow in Pharmacology
Dorothy Kibler	Fellow in Pharmacology

This course is designed to include those phases of pharmacology necessary for an intelligent use of drugs in the treatment of disease. The didactic instruction includes materia medica, pharmacy, prescription-writing, toxicology, posology, pharmacodynamics, and experimental therapeutics. The laboratory exercises parallel the course of lectures.

In addition, optional conference periods and lectures are available for students desiring further instruction or advice.

Total hours: 192.

Graduate Courses. Consult the catalogue of the Graduate School for descriptions of the graduate courses offered by members of the staff.

DEPARTMENT OF PATHOLOGY

Hugh R. Spencer	Professor of Pathology
Robert B. Wright	Associate Professor of Pathology
C. Gardner Warner	Associate Professor of Pathology
Walter C. Merkel	Assistant Professor of Pathology
D. James Greiner	Assistant Professor of Pathology
Howard J. Maldeis	Associate in Pathology
Albert E. Goldstein	Associate in Pathology
Frank B. Kindell	Associate in Pathology
William S. Love, Jr.	Instructor in Pathology
Leon Freedom	Instructor in Pathology
Benjamin Abeshouse	Instructor in Pathology
Milton S. Sacks	Instructor in Pathology
Henry L. Wollenweber	Instructor in Pathology
Edward F. Cotter	Instructor in Pathology
Robert L. Graham	Instructor in Pathology
★Conrad B. Acton	Assistant in Pathology
John E. Savage	Assistant in Pathology
Harry C. Hull	Assistant in Pathology
Howard B. Mays	Assistant in Pathology
Ephraim T. Lisansky	Assistant in Pathology
Karl F. Mech	Assistant in Pathology
David Hollander	Assistant in Pathology
John A. Wagner	Weaver Fellow in Pathology

Courses of instruction in Pathology are given during the second and third years. The courses are based on the previous study of normal structure and function and aim to outline the history of disease. The relationship between clinical symptoms and anatomical lesions is constantly stressed.

General Pathology. *Second Semester, Second Year.* This course includes the study of disturbances of the body fluids; disturbances of structure, nutrition and metabolism of cells; disturbances of fat, carbohydrate and protein metabolism; disturbances of pigment metabolism; inflammation and tumors.

Laboratory instruction is based on the study of prepared slides (loan collection) and corresponding gross material.

Applied Pathology, Including Gross Morbid Anatomy and Morbid Physiology. *Third Year.* The laboratory instruction in this course is carried out in small teaching museums where prepared specimens and material from autopsies with clinical histories and sections are available for study. For this work the class is divided into small groups. Clinical correlation is stressed.

Autopsies. *Third Year.* Students in small groups attend autopsies at the morgues of the University Hospital and the Baltimore City Hospitals.

Clinical-Pathological Conference. (*Fourth Year.*) These exercises are held in collaboration with the Department of Medicine. Selected cases are discussed and autopsy findings are presented.

Second year	184 hours
Third year	160 hours
Fourth year	30 hours
Total	374 hours

DEPARTMENT OF MEDICINE

Maurice C. Pincoffs	Professor of Medicine
Jos. E. Gichner	Professor of Clinical Medicine and Physical Therapeutics
G. Carroll Lockard	Professor of Clinical Medicine
Harvey G. Beck	Professor of Clinical Medicine
Thomas R. Sprunt	Professor of Clinical Medicine
Paul W. Clough	Associate Professor of Medicine
Sydney R. Miller	Associate Professor of Medicine
Walter A. Baetjer	Associate Professor of Medicine
Wm. S. Love, Jr.	Associate Professor of Medicine
Raymond Hussey	Associate Professor of Medicine
Louis A. M. Krause	Associate Professor of Medicine
H. R. Peters	Associate Professor of Medicine
T. Nelson Carey	Associate Professor of Medicine
William H. Smith	Associate Professor of Clinical Medicine
H. J. Maldeis	Associate Professor of Medical Jurisprudence
George McLean	Assistant Professor of Medicine
John E. Legge	Assistant Professor of Medicine
Thomas C. Wolff	Assistant Professor of Medicine
H. M. Bubert	Assistant Professor of Medicine
J. S. Eastland	Assistant Professor of Medicine
Milton S. Sacks	Assistant Professor of Medicine
E. B. Freeman	Lecturer in Medicine
Charles R. Goldsborough	Lecturer in Medicine
W. N. Bispham, Col., M. C., U. S. A. (ret.)	Lecturer in Medicine
Wetherbee Fort	Associate in Medicine
Frank J. Geraghty	Associate in Medicine
L. P. Gundry	Associate in Medicine
E. H. Tonolla	Associate in Medicine
Harry V. Langeluttig	Associate in Medicine
Sol Smith	Associate in Medicine
Samuel T. Helms	Instructor in Medicine
Earl L. Chambers	Instructor in Medicine
David Tenner	Instructor in Medicine
R. B. Mitchell, Jr.	Instructor in Medicine

Samuel Legum	Instructor in Medicine
Robert W. Garis	Instructor in Medicine
M. S. Shiling	Instructor in Medicine
H. Edmund Levin	Instructor in Medicine
Robert A. Reiter	Instructor in Medicine
W. Grafton Hersperger	Instructor in Medicine
Meyer W. Jacobson	Instructor in Medicine
★Conrad Acton	Instructor in Medicine
★Hugh G. Whitehead	Instructor in Medicine
Philip D. Flynn	Instructor in Medicine
Edward S. Kallins	Instructor in Medicine
★Irving Freeman	Instructor in Medicine
John A. Myers	Instructor in Medicine
Kurt Levy	Instructor in Medicine
William G. Helfrich	Instructor in Medicine
Francis G. Dickey	Instructor in Medicine
★W. H. Triplett	Assistant in Medicine
Morris Fine	Assistant in Medicine
William H. Grenzer	Assistant in Medicine
George Silverton	Assistant in Medicine
Lawrence Katzenstein	Assistant in Medicine
Leon Ashman	Assistant in Medicine
Harry M. Robinson, Jr.	Assistant in Medicine
Nathaniel Beck	Assistant in Medicine
★Joseph M. Blumberg	Assistant in Medicine
Paul E. Carliner	Assistant in Medicine
Samuel Snyder	Assistant in Medicine
S. Edwin Muller	Assistant in Medicine
Louis J. Kroll	Assistant in Medicine
John L. Atkins	Assistant in Medicine
William K. Waller	Assistant in Medicine
Ephraim T. Lisansky	Assistant in Medicine
C. Edward Leach	Assistant in Medicine
Nachman Davidson	Assistant in Medicine
Richard N. Tillman	Assistant in Medicine
Samuel T. R. Revell, Jr.	Assistant in Medicine
Frederick J. Vollmer	Hitchcock Fellow in Medicine

GENERAL OUTLINE

Second Year

Introduction to clinical medicine.

(a) Introductory physical diagnosis.
    (1 hour a week, first semester; 2 hours a week, second semester.)
(b) Medical clinics.
    (1 hour a week, second semester.)

Third Year

I. The methods of examination (13 hours a week).
    (a) History taking.
    (b) Physical diagnosis.
    (c) Clinical pathology.

These subjects are taught and practiced in the hospital out-patient department and in the clinical laboratory.

II. The principles of medicine (200 hours).
  (a) Lectures, clinics and demonstrations in general medicine, neurology, pediatrics, psychiatry and preventive medicine.

III. The principles of therapeutics (15 hours).
  Lectures and demonstrations.

Fourth Year

The practice of medicine.

I. Clinical clerkship on the medical wards.
  (26 hours a week for ten weeks.)
  (a) Responsibility, under supervision, for the history, physical examination, laboratory examinations and progress notes of assigned cases.
  (b) Ward classes in general medicine, the medical specialties, and therapeutics.

II. Clinics in general medicine and the medical specialties.
  (6 hours a week.)

III. Dispensary work in the medical specialties.

IV. Clinical-pathological conferences (1 hour a week).

MEDICAL DISPENSARY WORK

The medical dispensaries of both the Mercy and the University Hospitals are utilized for teaching in the third year. Each student spends two hours daily for ten weeks in dispensary work. The work is done in groups of four to six students under an instructor. Systematic history-taking is especially stressed. Physical findings are demonstrated. The student becomes familiar with the commoner acute and chronic disease processes.

PHYSICAL DIAGNOSIS

Second Year. Didactic lectures and practical demonstrations in topographical anatomy and normal physical signs.

Third Year. The class is divided into small groups, and each section receives instruction for two hours daily for ten weeks. This course is given at the City Hospitals. The large clinical material there is utilized to give each student the opportunity to familiarize himself with the common types of bodily structure, with the normal variations in physical signs and with the physical signs of the chief pulmonary, circulatory and abdominal diseases.

A course of lectures (1 hour a week) on physical diagnosis supplements the practical work in this subject.

THERAPEUTICS

Third Year. General therapeutics and materia medica are taken up and an effort is made to familiarize the student with the practical treatment of disease. The special therapy of the chief diseases is then reviewed. One hour a week. Dr. Lockard.

Fourth Year. Special consideration is given to the practical application of therapeutic principles in bedside teaching and the chief therapeutic methods are demonstrated.

Students attend therapeutic ward rounds once a week throughout their medical trimester.

TUBERCULOSIS

During the third year in connection with the instruction in physical diagnosis a practical course is given at the Municipal Tuberculosis Hospital. Stress is laid upon the recognition of the physical signs of the disease, as well as upon its symptomatology and gross pathology.

CARDIOLOGY

During the fourth year an elective course in cardiology is offered at the Mercy Hospital. The course occupies one and one-half hours weekly. Physical diagnosis, electrocardiography and the therapeutic management of cardiac cases are stressed.

SYPHILIS

Third Year. During the third year the subject of syphilis is dealt with in the lecture course.

Fourth Year. An elective course in the therapeutic management of syphilis is offered in the dispensary.

CLINICAL PATHOLOGY

Milton S. Sacks	Assistant Professor of Medicine and Head of Department of Clinical Pathology
H. R. Peters	Associate Professor of Medicine
Samuel T. Helms	Instructor in Medicine
John A. Myers	Instructor in Medicine
★Hugh G. Whitehead	Assistant in Medicine
★Joseph M. Blumberg	Assistant in Medicine
Richard N. Tillman	Assistant in Medicine
S. Edwin Muller	Assistant in Medicine

During the third year the student is thoroughly drilled in the technic of the usual clinical laboratory work, so that he is able to perform all routine examinations which may be called for during his fourth year, in connection with the work in the wards and dispensary.

The practical work is supplemented by a series of didactic lectures and demonstrations in which the entire teaching staff of the department takes an active part. The microscopical and chemical study of blood, exudates and transudates, gastric juice, spinal fluid, feces and urine are successively taken up, and special attention directed to the clinical significance of the findings.

Clinical parasitology from the standpoint of the infecting agent and the carrier is given careful consideration.

The entire course is thoroughly practical. Each student provides his own microscope and blood counters and is supplied a haemoglobinometer for his exclusive use. Every two students are equipped with a special laboratory outfit for all routine purposes.

During the fourth year the student applies in the laboratories of the various affiliated hospitals what he has learned during the preceding year. He is also supplied with a laboratory outfit which is sufficiently complete to enable him to work independently of the general equipment. Special instructors are available during certain hours to give necessary assistance and advice.

GASTRO-ENTEROLOGY

Theodore H. Morrison	Clinical Professor of Gastro-Enterology
Maurice Feldman	Assistant Professor of Gastro-Enterology
Zachariah Morgan	Assistant Professor of Gastro-Enterology
Samuel Morrison	Assistant Professor of Gastro-Enterology
Joseph Sindler	Associate in Gastro-Enterology
Z. Vance Hooper	Associate in Gastro-Enterology
M. S. Koppelman	Instructor in Gastro-Enterology
Ernest Levi	Instructor in Gastro-Enterology
Albert J. Shochat	Instructor in Gastro-Enterology
Francis G. Dickey	Instructor in Medicine
John A. Myers	Assistant in Gastro-Enterology
William Greenfeld	Assistant in Gastro-Enterology
Nathaniel M. Beck	Assistant in Gastro-Enterology

Third Year. A series of six lectures is given on the diseases of the digestive tract.

Fourth Year. Clinics and demonstrations are given to the class for one hour a week; dispensary instruction to small groups throughout the entire session. Practical instruction is given in the use of modern methods of study of the diseases of the gastro-intestinal tract.

PSYCHIATRY

Ross McC. Chapman	Professor of Psychiatry
Ralph P. Truitt	Associate Professor of Psychiatry
Lawrence F. Woolley	Associate Professor of Psychiatry
★Harry Goldsmith	Assistant Professor of Psychiatry
H. W. Newell	Assistant Professor of Psychiatry
Harry M. Murdock	Assistant Professor of Psychiatry
Leslie B. Hohman	Lecturer in Psychiatry
★A. Russell Anderson	Instructor in Psychiatry
★Philip S. Wagner	Instructor in Psychiatry
Hans Loewald	Fellow in Psychiatry
Sidney Berman	Fellow in Psychiatry

First Year. The student attends six lectures dealing with the development and function of the normal personality.

Second Year. The student attends fourteen lectures dealing with psychopathology.

Third Year. Six lectures on psychopathology are given; twelve hours of lectures and demonstrations are devoted to reaction types; six hours of lectures and demonstrations to the psychoses; thirty hours to history-taking and the study of cases in the out-patient clinic.

Fourth Year. The neuroses, psychoneuroses and psychoses are dealt with in ten hours of lectures and demonstrations. In this year the class is divided into sections for clinical conferences on selected cases.

PEDIATRICS

C. Loring Joslin	Professor of Pediatrics
Edgar B. Friedenwald	Professor of Clinical Pediatrics
T. Campbell Goodwin	Associate Professor of Pediatrics
John H. Traband	Assistant Professor of Pediatrics
Albert Jaffe	Assistant Professor of Pediatrics
A. H. Finkelstein	Assistant Professor of Pediatrics
Frederick B. Smith	Assistant Professor of Pediatrics
J. Edmund Bradley	Assistant Professor of Pediatrics
Clewell Howell	Associate in Pediatrics
Samuel S. Glick	Associate in Pediatrics
F. Stratner Orem	Associate in Pediatrics
William M. Seabold	Associate in Pediatrics
Thomas J. Coonan	Associate in Pediatrics
G. Bowers Mansdorfer	Associate in Pediatrics
M. Paul Byerly	Instructor in Pediatrics
Jerome Fineman	Instructor in Pediatrics
B. Matthew Debuskey	Instructor in Pediatrics
Gibson J. Wells	Instructor in Pediatrics
W. J. Schmitz	Assistant in Pediatrics
Israel P. Meranski	Assistant in Pediatrics
H. Whitney Wheaton	Assistant in Pediatrics
Lauriston L. Keown	Assistant in Pediatrics
Israel Rosen	Assistant in Pediatrics
Robert J. Gore	Assistant in Pediatrics
Earle S. Scott	Assistant in Pediatrics

Third Year. The course is presented as follows:

Lectures on infant feeding and the fundamentals of diseases of infants and children. (15 hours.)

Lectures on contagious diseases in conjunction with the Department of Hygiene and Preventive Medicine. (14 hours.)

A special course in physical diagnosis is given at City Hospitals. (20 hours.)

Clinical conferences demonstrating diseases of the newly-born. (6 hours.)

Fourth Year. An amphitheatre clinic is given at which patients are shown to demonstrate the features of the diseases discussed. (30 hours.)

Conferences and demonstrations are given in problems concerning diagnosis, care, treatment and clinical pathology of the diseases of infants and children. (30 hours.)

Students are assigned subjects for the preparation of theses.

Clinical clerkships are assigned on the pediatric wards, where experience is gained in taking histories, making physical examinations, doing routine laboratory work, and following up the patients' progress. This is under the supervision of the visiting staff. (140 hours.)

Instruction is given in the pediatric clinic of the out-patient department of the University Hospital. This consists of 1½ hours daily for five weeks—30 minutes each day being devoted to a clinical demonstration of some interesting case by a member of the staff; one hour daily to taking histories and making physical examinations under supervision of one of the staff instructors. (45 hours.)

Total hours: 300.

NEUROLOGY

Irving J. Spear	Professor of Neurology
Andrew C. Gillis	Professor of Neurology
G. M. Settle	Associate Professor of Neurology and Clinical Medicine
Leon Freedom	Associate Professor of Neurology
Benjamin Pushkin	Assistant Professor of Neurology
Philip F. Lerner	Instructor in Neurology
Bernhard Badt	Instructor in Neurology
William L. Fearing	Instructor in Neurology
Charles E. Balfour	Assistant in Neurology
Edward F. Cotter	Assistant in Neurology
Harry B. Teitelbaum	Assistant in Neurology
John W. Machen	Assistant in Neurology

Second Year. Fifteen one-hour lectures are given to correlate the anatomy and physiology of the nervous system with clinical neurology.

Third Year. Twenty hours of instruction are given to the whole class in neuropathology supplemented with pathological demonstrations. Sixteen lecture-demonstrations are given in which the major types of the diseases of the nervous system are discussed. A course is also given at the Baltimore City Hospitals, consisting of six periods of two hours each, in which the students in small groups carry out complete neurological examinations of selected cases which illustrate the chief neurological syndromes.

Fourth Year. A clinical conference one hour each week is given to the whole class at the University and Mercy Hospitals. All patients presented at these clinics are carefully examined. Complete written records are made by the students who demonstrate the patients before the class. The patients are usually assigned one or two weeks before they are presented, and each student in the class must study and present one or more patients during the year.

Ward Class Instruction. Nine hours of instruction are given to each student in small sections at the University and Mercy Hospitals. In these classes the students come in close personal contact with the patients in the wards under the supervision of the instructor.

Dispensary Instruction. Small sections are instructed in the dispensaries of the University and Mercy Hospitals five afternoons each week. In this way students are brought into contact with nervous diseases in their early and late manifestations.

HYGIENE AND PUBLIC HEALTH

Huntington Williams	Professor of Hygiene and Public Health
William H. F. Warthen	Assistant Professor of Hygiene and Public Health
Ross Davies	Assistant Professor of Hygiene and Public Health
Myron G. Tull	Instructor in Hygiene and Public Health

Third Year. A one-hour lecture is given to the whole class each Monday from September to May. Basic instruction is given in the clinical and public health aspects of the communicable diseases. The lectures are under the auspices of the Department of Medicine and are given by staff members of that department, including physicians representing pediatrics and hygiene and public health.

Fourth Year. Two-hour instruction periods are devoted to the whole class, in groups of ten to fifteen students, on six Wednesday afternoons. These sessions enable the students themselves to prepare birth and death certificates, to vaccinate against smallpox and to conduct other practical public health procedures. In addition there are four Wednesday afternoon field inspection trips for each third of the class. These trips, under guidance of full time public health workers, include visits to (1) city water filtration plant, (2) sewage disposal plant, (3) milk pasteurization plant and bakery and (4) industrial plant which has an active program of hygiene. Elective case work is assigned the Western Health District.

The course deals with the fundamentals of public health and supplements the work in the third year. The major emphasis in both years is on the practice of preventive medicine and the relation of prevention to diagnosis and treatment. The whole class, in small groups, receives practical instruction at Sydenham Hospital, the one-hundred bed communicable disease hospital of the Baltimore City Health Department.

MEDICAL JURISPRUDENCE

Howard J. Maldeis	Associate Professor of Medical Jurisprudence Chief Medical Examiner, Maryland

Third Year. This course embraces a summary of some of the following: Proceedings in criminal and civil prosecution, medical evidence and testimony, identity and its general relations, personal identity, rape, criminal abortions, signs of death, wounds in their medico-legal relations, natural and homocidal death, malpractice, insanity, and medico-legal autopsies.

Total hours: 3.

DEPARTMENT OF SURGERY

Arthur M. Shipley	Professor of Surgery
Walter D. Wise	Professor of Surgery
Page Edmunds	Professor of Traumatic Surgery
Charles Bagley, Jr.	Professor of Neurological Surgery
Elliott H. Hutchins	Professor of Clinical Surgery
Charles Reid Edwards	Professor of Clinical Surgery
F. L. Jennings	Professor of Clinical Surgery
Thomas B. Aycock	Professor of Clinical Surgery
D. J. Pessagno	Clinical Professor of Surgery
Monte Edwards	Clinical Professor of Surgery
Thomas R. Chambers	Associate Professor of Surgery
R. W. Locher	Associate Professor of Clinical Surgery
Edward S. Johnson	Associate Professor of Surgery
Charles A. Reifschneider	Associate Professor of Traumatic Surgery
N. Clyde Marvel	Associate Professor of Surgery

Richard G. Coblentz	Associate Professor of Neurological Surgery
Grant E. Ward	Associate Professor of Surgery
Cyrus F. Horine	Associate Professor of Surgery
George H. Yeager	Assistant Professor of Surgery
H. F. Bongardt	Assistant Professor of Surgery
Henry F. Ullrich	Assistant Professor of Surgery
I. O. Ridgley	Associate in Surgery
C. W. Peake	Associate in Surgery
Edward A. Kitlowski	Associate in Plastic Surgery
James W. Nelson	Associate in Surgery
I. Ridgeway Trimble	Associate in Surgery
Richard T. Shackelford	Associate in Surgery
Simon H. Brager	Associate in Surgery
Raymond F. Helfrich	Associate in Surgery
Harry C. Hull	Associate in Surgery
Otto C. Brantigan	Associate in Surgery
William R. Johnson	Instructor in Surgery
E. M. Hanrahan	Instructor in Surgery
S. Demarco	Instructor in Surgery
★Karl J. Steinmueller	Instructor in Surgery
Luther E. Little	Instructor in Surgery
J. Frank Hewitt	Instructor in Surgery
Murray M. Copeland	Instructor in Surgery
E. Eugene Covington	Instructor in Surgery
J. Duer Moores	Instructor in Surgery
Thurston R. Adams	Instructor in Surgery
Arthur G. Siwinski	Instructor in Surgery
William R. Geraghty	Assistant in Surgery
Howard B. McElwain	Assistant in Surgery
A. V. Buchness	Assistant in Surgery
T. J. Touhey	Assistant in Surgery
Clyde F. Karns	Assistant in Surgery
Samuel H. Culver	Assistant in Surgery
Albert R. Wilkerson	Assistant in Surgery
L. T. Chance	Assistant in Surgery
Samuel McLanahan, Jr.	Assistant in Surgery
W. Allen Deckert, Jr.	Assistant in Surgery
★Samuel E. Proctor	Assistant in Surgery
George Govatos	Assistant in Surgery
Dwight Currie	Assistant in Surgery
Robert F. Healy	Assistant in Surgery
Herbert M. Foster	Assistant in Surgery
Daniel R. Robinson	Assistant in Surgery
Robert F. Chenowith	Assistant in Surgery
Joseph V. Jerardi	Assistant in Surgery
Eldred Roberts	Assistant in Surgery
Donald B. Grove	Assistant in Surgery
★William S. Cheney	Assistant in Surgery
Herbert E. Reifschneider	Assistant in Surgery
George H. Brouillet	Assistant in Surgery

Harry C. Bowie	Assistant in Surgery
William M. Garlick	Assistant in Surgery

Instruction is given by means of lectures, laboratory work, recitations, dispensary work, bedside instruction, ward classes, and clinics. The work begins in the second year, and continues throughout the third and fourth years.

The teaching is done in the anatomical laboratory, operative surgery laboratory, the dispensaries, wards, clinical laboratories and operating rooms of the University and Mercy Hospitals, and in the wards and operating rooms of the Baltimore City Hospitals.

SECOND YEAR

Topographic and Surgical Anatomy. The course is designed to bridge the gap between anatomy in the abstract and clinical anatomy as applied to the study and practice of medicine and surgery.

The teaching is done in the anatomical laboratory, and students are required to demonstrate all points, outlines, and regions on the cadaver. Underlying regions are dissected to bring out outlines and relations of structures.

Two lectures and two laboratory periods per week during the second semester. Drs. Brantigan, Hull, Adams, Brouillet, H. E. Reifschneider and Bowie.

Total hours: 96.

Principles of Surgery. This course includes history-taking, records of physical examinations, and of operations and progress notes; the preparation of surgical dressings, suture materials and solutions. It includes inflammation, infections, ulcers, gangrene, fistulae and sinuses, hemorrhage, shock and tumors. Lectures and conferences, two hours per week for one semester, are given to the whole class. Dr. Aycock.

THIRD YEAR

General and Regional Surgery. Lectures, recitations and clinics on the principles of surgery and general surgery are given three hours a week to the whole class. Dr. C. R. Edwards.

The class is divided into groups and receives instruction in history-taking, gross pathology, and surgical diagnosis—at the bedside and in the dead-house of the Baltimore City Hospitals. Drs. Aycock, C. A. Reifschneider, Toulson, Hull, Yeager and Brantigan.

Operative Surgery. Two courses are given in operative surgery under the supervision of Dr. Yeager assisted by Drs. Peake, Ullrich, Brager, Brouillet, Chenowith, Culver, Deckert, Foster, Govatos, Healy, Jerardi, Karns, Mech and Williamson. The class is divided into sections and each section is given practical and individual work under the supervision of the instructors.

Fractures and Dislocations. This course consists of instruction in the various forms of fractures, dislocations and their treatment. There is a regular schedule of didactic lectures, which is supplemented by practical demonstrations in diagnosis and treatment.

Surgical Dispensary. Under supervision, the student takes the history, makes the physical examinations, attempts the diagnosis, and, as far as possible, carries out the treatment of the ambulatory surgical patients in the University and in the Mercy Hospitals. Mercy Hospital—Dr. Helfrich assisted by the whole dispensary staff. University Hospital—Dr. Yeager assisted by the whole dispensary staff.

FOURTH YEAR

CLINICS. A weekly clinic is given at the Mercy and at the University Hospitals to one-half the class throughout the year. As far as possible this is a diagnostic clinic. Mercy Hospital—Dr. Wise. University Hospital—Dr. Shipley.

SURGICAL PATHOLOGY A weekly exercise of one hour at Mercy Hospital for one semester at which specimens from the operating room and museum are studied in the gross and microscopically in relation to the case history. Dr. Pessagno.

TRAUMATIC SURGERY. This course deals with operative and post-operative treatment of accident cases, with instructions as to the relationship between the state, the employee, the employer, and the physician's duty to each. One hour a week to sections of the class throughout the year. Dr. Edmunds.

CLINICAL CLERKSHIP. This work includes the personal study of assigned hospital patients, under supervision of the staffs of the University and Mercy Hospitals, history-taking, and physical examination of patients, laboratory examinations, attendance at operations and observation of post-operative treatment.

WARD CLASSES. Ward-class instruction in small groups will consist of ward rounds, surgical diagnosis, treatment and the after-care of operative cases. Mercy Hospital—Drs. Wise, Hutchins, Jennings, Pessagno and Marvel. University Hospital—Drs. Shipley, Edmunds, Edwards and Aycock.

ANAESTHESIA

S. GRIFFITH DAVIS	Professor of Anaesthesia
E. HOLLISTER DAVIS	Assistant in Anaesthesia
MARY J. O'BRIEN	Anaesthetist
RUTH ELLIOTT	Anaesthetist

THIRD YEAR

Lectures are given on the general physiology of anaesthesia, with consideration of special physiology of each anaesthetic agent; methods of induction and administration of anaesthesia; factors influencing the selection of the anaesthetic and types of anaesthetic agents; and the preparation and care of the anaesthetized patient.

The lectures are correlated with practical demonstrations during operative clinics at the City Hospitals.

FOURTH YEAR

During operative clinics in both surgery and gynecology each student will be given practical instruction in the administration of anaesthetics and will be required to record such changes as take place in blood pressure, pulse and respiration.

DERMATOLOGY

Harry M. Robinson	Professor of Dermatology
Francis Ellis	Assistant Professor of Dermatology
John R. Abercrombie	Associate in Dermatology
Harold M. Goodman	Associate in Dermatology
Harry M. Robinson, Jr.	Associate in Dermatology
★Lester N. Kolman	Instructor in Dermatology
Rollin C. Hudson	Assistant in Dermatology
Jeannette R. Heghinian	Assistant in Dermatology
Mark Hollander	Assistant in Dermatology
★Benjamin Highstein	Assistant in Dermatology

A weekly clinic is given at University and Mercy Hospitals throughout the year. This course consists of demonstrations of the common diseases of the skin, and conferences, in addition to a number of lectures on the general principles of dermatology. Drs. Robinson and Ellis.

Dispensary instruction is given in the diagnosis and treatment of skin diseases at the University Hospital by Dr. Robinson and dispensary staff, and at Mercy Hospital by Dr. Ellis and dispensary staff.

Third year	15 hours
Fourth Year	49 hours
Total	64 hours

ORTHOPAEDIC SURGERY

Allen Fiske Voshell	Professor of Orthopaedic Surgery
Albertus Cotton	Professor of Orthopaedic Surgery
Compton Riely	Clinical Professor of Orthopaedic Surgery
Moses Gellman	Associate Professor of Orthopaedic Surgery
Harry L. Rogers	Associate Professor of Orthopaedic Surgery
Henry F. Ullrich	Assistant Professor of Orthopaedic Surgery
I. H. Maseritz	Instructor in Orthopaedic Surgery
John V. Hopkins	Assistant in Orthopaedic Surgery

Didactic, clinical, bedside and out-patient instruction is given to the fourth year at the University and Mercy Hospitals and Dispensaries, Kernan Hospital for Crippled Children at Dickeyville and Baltimore City Hospitals. Instruction is also given to the third year in small groups at the Baltimore City Hospitals.

Weekly lectures throughout the year present all phases of orthopaedic surgery except fractures; brief discussions and demonstrations of physical therapy are included.

Fourth year groups are given more intimate instruction biweekly at one of the above institutions; fracture cases are included here.

Third year	60 hours
Fourth year	90 hours
Total	150 hours

ROENTGENOLOGY

‡Henry J. Walton	Professor of Roentgenology
Walter L. Kilby	Acting Professor of Roentgenology
Albertus Cotton	Professor of Roentgenology
Eugene L. Flippin	Associate in Roentgenology

During the academic year small groups of the fourth year class are given weekly instruction in the diagnostic and therapeutic uses of the Roentgen rays. An effort is made to familiarize the student with the indications for and limitations of Roentgen ray examinations. The history, physics and practical application of Roentgen rays are alluded to but not stressed. Conferences are held with the various departments during the school year which are also open to members of the fourth year class.

Total hours: 96.

DISEASES OF THE NOSE AND THROAT

Edward A. Looper	Professor of Diseases of the Nose and Throat
Waitman F. Zinn	Clinical Professor of Diseases of the Nose and Throat
Franklin B. Anderson	Associate Professor of Diseases of the Nose and Throat
W. Raymond McKenzie	Associate in Diseases of the Nose and Throat
Thomas R. O'Rourk	Associate in Diseases of the Nose and Throat
Cleo D. Stiles	Associate in Diseases of the Nose and Throat
Benjamin S. Rich	Associate in Diseases of the Nose and Throat
F. A. Kayser	Assistant in Diseases of the Nose and Throat
Theodore A. Schwartz	Assistant in Diseases of the Nose and Throat

Third Year. Instruction to whole class is given in the common diseases of the nose and throat, attention being especially directed to infections of the accessory sinuses, the importance of focal infections in the etiology of general diseases and modern methods of diagnosis. Lectures illustrated by lantern slides are given one hour weekly for seven weeks by Dr. Looper.

Fourth Year. Dispensary instruction is given for one and one-half hours daily, to small sections at the University and the Mercy Hospitals. The student is afforded an opportunity to study, diagnose and treat patients under supervision. Ward classes and clinical demonstrations are given in periods of one and one-half hours weekly throughout the session in the University and the Mercy Hospitals.

The Looper Clinic for bronchoscopy and esophagoscopy, recently established in the University Hospital, affords unusual opportunities for students to study diseases of the larynx, bronchi and esophagus. The clinic is open to students daily from 2 to 4 P.M., under direction of Dr. Looper.

The Mercy Hospital clinic for bronchoscopy and esophagoscopy is under the direction of Dr. Zinn. In these two clinics the etiology, symptomatology, diagnosis and treatment of foreign bodies in the air and food passages, as well as bronchoscopy, are taught to students, as an aid in the diagnosis and treatment of diseases of the lungs.

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‡ June 30, 1941.

Third year	9 hours
Fourth year	53 hours
Total	62 hours

GENITO-URINARY SURGERY

W. H. Toulson	Professor of Genito-Urinary Surgery
Kenneth D. Legge	Clinical Professor of Genito-Urinary Surgery
Austin H. Wood	Associate in Genito-Urinary Surgery
L. J. Millan	Associate in Genito-Urinary Surgery
L. K. Fargo	Associate in Genito-Urinary Surgery
John F. Hogan	Associate in Genito-Urinary Surgery
W. A. H. Councill	Associate in Genito-Urinary Surgery
★Harry S. Shelley	Associate in Genito-Urinary Surgery
Francis W. Gillis	Associate in Genito-Urinary Surgery
Samuel T. Helms	Instructor in Genito-Urinary Surgery
Howard B. Mays	Instructor in Genito-Urinary Surgery
Hugh Jewett	Assistant in Genito-Urinary Surgery

Third Year. This course is given for seven hours to the whole class. It consists of lectures and demonstrations, including the use of lantern slides and motion pictures. Dr. Toulson.

Fourth Year. The course in this year includes explanations and demonstrations of urethroscopy, cystoscopy, ureteral catheterization, renal function tests, urography, urine cultures and the various laboratory procedures. The teaching consists of clinics and ward rounds to small groups, and attendance by members of the senior class upon the out-patients in the dispensary. The student here is placed much on his own responsibility in arriving at a diagnosis. Members of the staff are in constant attendance for consultations. These dispensary classes are conducted at both the Mercy and University Hospitals where practically every variety of urogenital disease is seen and used for teaching purposes.

Third year	8 hours
Fourth year	64 hours
Total	72 hours

DISEASES OF THE RECTUM AND COLON

Charles F. Blake	Professor of Diseases of Rectum and Colon
J. Dawson Reeder	Professor of Diseases of Rectum and Colon
Monte Edwards	Clinical Professor of Diseases of Rectum and Colon
James C. Owings	Instructor in Diseases of the Rectum and Colon

Third Year. Seven lectures are given to the whole class. This course is for instruction in the diseases of the colon, sigmoid flexure, rectum and anus, and covers the essential features of the anatomy and physiology of the large intestine as well as the various diseases to which it is subject. Dr. Reeder and Dr. Edwards.

Fourth Year. Ward and dispensary instruction is given in the University and

Mercy Hospitals, where different phases of the various diseases are taught by direct observation and examination. The use of the proctoscope and sigmoidoscope in the examination of the rectum and sigmoid is made familiar to each student. Mercy Hospital—Dr. Blake. University Hospital—Drs. Reeder and Monte Edwards.

Third year	7 hours
Fourth year	16 hours
Total	23 hours

OTOLOGY

J. W. Downey	Professor of Otology
Franklin B. Anderson	Associate Professor of Otology
Benjamin S. Rich	Associate in Otology
Thomas R. O'Rourk	Associate in Otology
William A. Parr	Assistant in Otology
Cleo D. Stiles	Assistant in Otology

The course in otology is planned to give a practical knowledge of the anatomy and physiology of the ear, and its proximity and relationship to the brain and other vital structures. The inflammatory diseases, their etiology, diagnosis, treatment and complications are particularly stressed, with emphasis upon their relationship to the diseases of children, head-surgery and neurology.

Third Year. The whole class is given instruction by means of talks, anatomical specimens and lantern slides.

Fourth Year. Small sections of the class receive instruction and make personal examinations of patients under the direction of an instructor. The student is urged to make a routine examination of the ear in his ward work in general medicine and surgery.

Third year	12 hours
Fourth year	40 hours
Total	52 hours

NEUROLOGICAL SURGERY

Charles Bagley, Jr.	Professor of Neurological Surgery
Richard G. Coblentz	Associate Professor of Neurological Surgery

Third Year. The course covers instruction in diagnosis and treatment of surgical conditions of the brain, spinal cord and the peripheral nerves. Dr. Bagley.

Fourth Year. Weekly ward rounds and conferences are given at the University Hospital. Drs. Bagley and Coblentz.

Third year	12 hours
Fourth year	30 hours
Total	42 hours

ONCOLOGY

J. Mason Hundley, Jr.	Professor of Gynecology
Grant E. Ward	Associate Professor of Surgery
James W. Nelson	Associate in Surgery
Beverley C. Compton	Associate in Gynecology
John C. Dumler	Associate in Gynecology
Murray M. Copeland	Instructor in Surgery
E. Eugene Covington	Instructor in Surgery
J. Duer Moores	Instructor in Surgery
Ernest I. Cornbrooks, Jr.	Instructor in Gynecology
Arthur G. Siwinski	Instructor in Surgery
Eldred Roberts	Assistant in Surgery
Everett S. Diggs	Assistant in Gynecology

Every facility for the diagnosis and treatment of neoplastic diseases is available; this includes electro-surgery, radium therapy and deep X-ray therapy.

An out-patient clinic is held twice weekly which affords an opportunity for instruction to a limited number of students. The gynecological problems are under the supervision of Dr. Hundley, and the general surgical conditions are under the direction of Dr. Ward.

Instruction, other than dispensary teaching, is given to small groups of students, for one hour a week, in the history, physics and practical application of radium. Drs. Ward and Hundley.

Third year	8 hours
Fourth year	16 hours
Total	24 hours

ORAL SURGERY

Brice M. Dorsey	Professor of Oral Surgery
Charles A. Reifschneider	Associate Professor of Oral Surgery
Grant E. Ward	Associate Professor of Oral Surgery
J. Herbert Wilkerson	Assistant Professor of Oral Surgery
Herbert E. Reifschneider	Instructor in Oral Surgery
William E. Hahn	Instructor in Oral Surgery
Conrad L. Inman	Instructor in Oral Surgery
Vernon D. Kaufman	Instructor in Oral Surgery
B. Wallace Inman	Instructor in Oral Surgery
Dorsey R. Tipton	Instructor in Oral Surgery

This section in the Department of Surgery is established for the teaching of both medical and dental students. A new subdivision in the dispensary has also been established. Beds will be provided in the University Hospital for the care of patients who will be available for the teaching of students from both schools.

Senior year: clinics weekly.

Ward instruction and group teaching are given. This includes diagnosis and treatment of diseases of the face, mouth and jaws.

INDUSTRIAL MEDICINE AND SURGERY

Page Edmunds	Professor of Traumatic Surgery
G. Carroll Lockard	Professor of Clinical Medicine
Charles A. Reifschneider	Associate Professor of Traumatic Surgery and Oral Surgery
Thurston R. Adams	Instructor in Surgery

This section is under the combined supervision of the medical and surgical departments. It is a cooperative effort by members of the medical school and hospital staff to afford means for clinical and laboratory study of the patient who has been subjected to traumatic or medical industrial hazard, so that adequate care may be instituted to promote his physical well-being. The facilities of the laboratories of the medical school and hospital are available as required.

Under direction of this department limited undergraduate instruction is given, especially in the methods of examination and of keeping records; and in the general medico-legal principles as they affect the industrial employee, the employer, the general insurers, the physician and the hospital. There is also instruction on methods of making life insurance and other physical examinations, whether for employment or for health purposes. The wards of the University, Mercy and City Hospitals provide for bed-side instruction.

Total hours: 48.

PLASTIC SURGERY

Edward A. Kitlowski	Associate in Plastic Surgery

This course is designed to acquaint students with the problems of reconstructive and plastic surgery. A subdivision in the dispensary has been established and beds for patients will be available for instruction in this course at the University and City Hospitals and Kernan's Hospital for Crippled Children.

Third Year. Five lectures are given to the whole class. Dispensary instruction is provided on Tuesdays and Fridays.

Fourth Year. Ward rounds and operative demonstrations are held at the hospitals.

DEPARTMENT OF OBSTETRICS

L. H. Douglass	Professor of Obstetrics
J. McFarland Bergland	Associate Professor of Obstetrics
Emil Novak	Associate Professor of Obstetrics
J. G. M. Reese	Assistant Professor of Obstetrics
M. Alexander Novey	Assistant Professor of Obstetrics
Isadore A. Siegel	Assistant Professor of Obstetrics
John E. Savage	Associate in Obstetrics
Margaret B. Ballard	Associate in Obstetrics
Dudley P. Bowe	Associate in Obstetrics
Frank K. Morris	Instructor in Obstetrics
Hugh B. McNally	Instructor in Obstetrics
Kenneth B. Boyd	Assistant in Obstetrics
W. Allen Deckert	Assistant in Obstetrics

Jaroslav Hulla	Assistant in Obstetrics
Marius P. Johnson	Assistant in Obstetrics
Maxwell L. Mazer	Assistant in Obstetrics
★Joseph M. Blumberg	Assistant in Obstetrics
Ferd. E. Kadan	Assistant in Obstetrics
J. Edward Norris	Assistant in Obstetrics
Jacob R. Jensen	Assistant in Obstetrics
★J. Warren Albrittain	Assistant in Obstetrics
D. McClellen Dixon	Assistant in Obstetrics
★W. Kenneth Mansfield, Jr.	Assistant in Obstetrics
Bernard W. Donohue	Assistant in Obstetrics
George H. Davis	Assistant in Obstetrics

Third Year. The lectures and recitations consisting of three hours teaching weekly are designed to cover the anatomy of the female generative tract and the bony pelvis, the physiology and development of the ovum, and the physiology of pregnancy and labor. Following this the pathology of pregnancy, labor and the puerperium are taken up. Drs. Douglass, Novak, Reese, Novey, Siegel and Savage.

Each student spends time during his junior year at the Baltimore City Hospitals observing, assisting and finally delivering patients under strict supervision. Each student sees about twenty deliveries there, and does a considerable amount of the routine work.

The junior students are assigned as assistants to the seniors in the home delivery service and accompany them on deliveries.

Each student receives, in small groups, ten hours instruction in palpation of patients and mensuration of the pelvis and demonstrations of the mechanism of labor. Drs. Siegel and McNally.

Fourth Year. At the weekly clinical conference, cases are presented and discussed and the student body is encouraged to offer opinions and to ask questions. There is no didactic teaching done, and an earnest effort is made to keep it, in every sense of the word, a conference. Dr. Douglass.

The ward classes are held three times weekly for five weeks for each group. Various subjects are assigned and discussed, patients and their histories are presented. Drs. Douglass, Reese and Novey.

Manikin instruction is given once a week. Drs. Morris, Bowe and Dixon.

During the same five-week period, the students are sent into patients' homes to conduct deliveries under supervision of a senior member of the house staff and with the assistance of a graduate nurse. The student is held responsible for the complete conduct of each assigned case.

Each student spends thirty hours in the prenatal clinic, taking histories and examining patients under supervision.

Finally, the students are invited to attend the monthly meetings of The Committee on Maternal Mortality, where all maternal deaths occurring in Baltimore are openly discussed.

Third year	148 hours
Fourth year	102 hours
Total	250 hours

DEPARTMENT OF GYNECOLOGY

J. Mason Hundley, Jr.	Professor of Gynecology
Abram S. Samuels	Clinical Professor of Gynecology
Thomas K. Galvin	Assistant Professor of Gynecology
Leo Brady	Assistant Professor of Gynecology
R. G. Willse	Assistant Professor of Gynecology
George A. Strauss, Jr	Assistant Professor of Gynecology
Edward P. Smith	Assistant Professor of Gynecology
John T. Hibbitts	Associate in Gynecology
Kenneth B. Boyd	Associate in Gynecology
John C. Dumler	Associate in Gynecology
Beverley C. Compton	Associate in Gynecology
Houston Everett	Associate in Gynecology
Thomas S. Bowyer	Instructor in Gynecology
Joseph V. Castagna	Instructor in Gynecology
Ernest S. Edlow	Instructor in Gynecology
W. Allen Deckert	Instructor in Gynecology
Frank K. Morris	Instructor in Gynecology
Ernest I. Cornbrooks, Jr.	Instructor in Gynecology
William B. Settle	Instructor in Gynecology
H. L. Granoff	Assistant in Gynecology
J. J. Erwin	Assistant in Gynecology
Everett S. Diggs	Assistant in Gynecology
William K. Diehl	Assistant in Gynecology

Third Year. A course of thirty lectures and recitations is given to the whole class. In addition, a short course of lecture-demonstrations is given at the Baltimore City Hospitals, consisting of eight periods of one hour each, in which small groups of students are instructed in the fundamentals of gynecological diagnosis and examination.

Fourth Year. Operative clinics—lectures and demonstrations—are given six hours per week, for five weeks, to sections of the class.

Instruction in female urology is given. A small number of students may attend the cystoscopic dispensary which is held twice weekly.

The course in gynecology also includes instruction in the diagnosis and treatment of cancer of the generative organs. Small groups of students attend the oncological dispensary for additional work.

Third year	38 hours
Fourth year	74 hours
Total	112 hours

DEPARTMENT OF OPHTHALMOLOGY

Clyde A. Clapp	Professor of Ophthalmology
M. Randolph Kahn	Clinical Professor of Ophthalmology
H. K. Fleck	Clinical Professor of Ophthalmology
Jonas Friedenwald	Lecturer in Ophthalmic Pathology
R. D. West	Associate in Ophthalmology
Henry F. Graff	Associate in Ophthalmology

Joseph I. Kemler	Associate in Ophthalmology
F. A. Holden	Instructor in Ophthalmology
F. Edwin Knowles, Jr.	Instructor in Ophthalmology
John G. Runkle	Assistant in Ophthalmology
Thomas R. O'Rourk	Assistant in Ophthalmology
Jerome Snyder	Assistant in Ophthalmology
Milton C. Lang	Assistant in Ophthalmology

Third Year. Second semester. Dr. Kahn reviews the anatomy and physiology of the eye and discusses the methods used in making the various examinations. Errors of refraction and their effect upon the general system are explained. Weekly section work, demonstrating the use of the ophthalmoscope, is carried on during the entire session.

Fourth Year. Clinics and demonstrations are given in diseases of the eye, weekly, for one year. Dr. Clapp.

This course consists of lectures upon the diseases of the eye, with particular reference to their diagnosis and relation to general medicine. Special lectures will be given upon vascular changes in the eye and upon the pathology of the eye. Some operations will be demonstrated by motion pictures.

Weekly ward classes are held at the University, The Baltimore Eye, Ear and Throat and Mercy Hospitals during which the eye grounds in the various medical and surgical conditions are demonstrated. Also daily demonstrations are given in the taking of histories and the diagnosis and treatment of the various conditions as seen in the dispensary. Drs. Fleck, West, Kemler, Graff and Knowles.

Third year	20 hours
Fourth year	104 hours
Total	124 hours

HISTORY OF MEDICINE

Louis A. M. Krause	Associate Professor of Medicine

Beginning with this session a group of lectures on the history of medicine will be presented on selected phases and trends of the development of medical knowledge and practice. It is planned to avoid duplication of subject matter for at least four years.

These lectures are offered primarily for our students, but a cordial invitation is extended to anyone who may wish to attend.

Announcement of the lectures will be made by mail and on the bulletin board of the School of Medicine.

ART AS APPLIED TO MEDICINE

Carl Dame Clarke	Associate Professor of Art as Applied to Medicine

This department is maintained for the purpose of supplying pictorial and plastic illustrations for visual teaching in the classrooms of the medical school and for publication in scientific periodicals.

Special courses of instruction are given to qualified students.

FIRST YEAR SCHEDULE

FIRST SEMESTER, SEPTEMBER 25, 1941 TO JANUARY 31, 1942

Hours	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
9.00 to 1.00	Gross Anatomy—Lectures and Laboratories *Bressler 1*					(9–12)
1.00 to 2.00	Lunch					
2.00 to 5.00	*Histology and Embryology *Bressler 2*			Histology and Embryology *Bressler 2*		

* Course begins October 9, 1941.

SECOND SEMESTER, FEBRUARY 2 TO MAY 30, 1942

Hours	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
9.00 to 12.00	Laboratory — Biological Chemistry *Sect. A*	Laboratory — Biological Chemistry *Sect. B*	Laboratory — Biological Chemistry *Sect. A*	Laboratory — Biological Chemistry *Sect. B*	10.30 to 11.30 Psychiatry *Adm. 1*	
12.00 to 12.50	Lunch	Lunch	Lunch	Lunch	Lunch	
12.50 to 1.50	Biological Chemistry *Adm. 1*	Biological Chemistry *Adm. 1*	Biological Chemistry *Adm. 1*	Biological Chemistry *Adm. 1*	Biological Chemistry *Adm. 1*	
2.00 to 5.00	*Neuro-anatomy *Bressler 2*	(Lecture, 2–3) Neuro-anatomy *Bressler 2*	Biological Chemistry Conference *Adm. 1*	(Lecture, 2–3) Neuro-anatomy *Bressler 2*	Biological Chemistry Conference *Adm. 1*	

* Course begins March 2, 1942.

Locations of Lecture Halls and Laboratories:

Adm. 1—First Floor, Administration Building, Lombard and Greene Streets.
A. H.—Anatomical Hall—Upper Hall, N. E. Cor. Lombard and Greene Streets.
C. H.—Chemical Hall, Lower Hall, N. E. Cor. Lombard and Greene Streets.
Biological Chemistry Laboratory—Third Floor, 31 South Greene Street.
Bressler Research Laboratory—29 S. Greene Street.
  Gross Anatomy—First Floor
  Histology and Embryology—Second Floor.
  Neuro-anatomy—Second Floor.

Mid-Year Examinations—January 26–31, 1942
Final Examinations—May 25–30, 1942

SECOND YEAR SCHEDULE

FIRST SEMESTER, SEPTEMBER 25, 1941 TO JANUARY 31, 1942

Hours	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
8.30 9.30	Physiology *Bressler 2*	Physiology *Bressler 2*	Medicine *Bressler 2*	Physiology *Bressler 2*	Physiology *Bressler 2*	
9.30 to 10.30	Physiology Conference *Bressler 2*	Bacteriology *Adm. 1*	Physiology *Bressler 2*	Pharmacology *Bressler 2*	Pharmacology *Bressler 2*	
10.30 to 12.30	†Bacteriology Laboratory				Psychiatry *C. H.* Neurological Diagnosis *C. H.*	
12.30	Lunch					
1.00 to 5.00	Pharmacology Lecture (1–2) *Bressler 2* *B* Physiology *A*	*A* Laboratory *B*	Elective	Pharmacology Laboratory (1 to 4) *B* Physiology *A*	*A* Laboratory *B*	

† Bacteriology Laboratory—Section work during the last month.

SECOND SEMESTER, FEBRUARY 2 TO MAY 30, 1942

Hours	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
8.30 to 9.30	Surgery *Bressler 2*	Surgery *Bressler 2*	Surgical Anatomy *Bressler 2*	Physical Diagnosis *Bressler 2*	Physical Diagnosis *Bressler 2*	
9.30 to 10.30	Pharmacology *Bressler 2*	Pharmacology *Bressler 2*	Surgical Anatomy Laboratory *Bressler 1*	Medical Clinic *Amp.*	Pharmacology *Bressler 2*	
10.30 to 11.30	Pathology *C. H.*	Pathology *C. H.*		Pathology *C. H.*	Pathology *C. H.*	
11.30	Lunch					
12.00 to 2.00	Pathology Laboratory	Pathology Laboratory	Immunology Laboratory	Pathology Laboratory	Pathology Laboratory	
2.00 to 3.00	Surgical Anatomy *Bressler 2*	‖Immunology Laboratory		Pharmacology Laboratory *Sect. A*	Pharmacology Laboratory *Sect. B*	
3.00 to 5.00	Surgical Anatomy Laboratory *Bressler 1*		Optional period Pathology Immunology	Physical Diagnosis *Sect. B* (3.00–5.00) *U. H. D.*	Physical Diagnosis *Sect. A* (3.00–5.00) *U. H. D.*	

‖ Immunology Laboratory—Section work during last two months.

Locations of Lecture Halls and Laboratories:

Adm. 1—First floor, Administration Building, Lombard and Greene Streets.
C. H.—Chemical Hall, Lower Hall, Lombard and Greene Streets.
Amp.—Wilson Memorial Amphitheatre, New University Hospital, Greene and Redwood Streets, Eighth Floor
U. H. D —University Hospital Dispensary, Old Hospital Building.

Laboratories:

Physiology, Pharmacology, Surgical Anatomy—Bressler Building.
Bacteriology, Immunology, Pathology, Second Floor, 31 S. Greene Street.

Mid-Year Examinations—January 26–31, 1942
Final Examinations— May 25–30, 1942

THIRD YEAR SCHEDULE

SEPTEMBER 25, 1941 TO MAY 30, 1942

SCHEDULE 1

Hours	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
8.30 to 9.30	(Whole Class) Obstetrics *C. H.* †Gynecology Apr. 20 to May 11	(Whole Class) Surgery *C. H.*	(Whole Class) Obstetrics *C. H.* †Gynecology Apr. 22 to May 13	(Whole Class) Surgery *C. H.*	(Whole Class) Pathology *C. H.*	(Whole Class) Surgery *C. H.* †Gynecology Feb. 7 to Mar. 28 inc.
9.30 to 10.00	Transfer to Baltimore City Hospitals					
10.00 to 12.00	Physical Diagnosis, Pathology, Neurology and Pediatrics at B. C. H.					
12.00 to 1.00	Transfer and Lunch	Transfer and Lunch	Lunch	Transfer and Lunch	Lunch	
1.00 to 2.00	(Whole Class) Otology, Proctology, Urology, Nose & Throat, Plastic Surgery *C. H.*	(Whole Class) *Gynecology †Eye—10 wks. (Feb. 3 to April 7) †Oncology —5 wks. (April 14 to May 12) *C. H.*	Medical Clinic *B. C. H.*	(Whole Class) Clinical Pathology *Bressler 2*	Obstetrical Clinic *B. C. H.*	
2.00 to 4.00	(Whole Class) Pathology Laboratory *31*		Surgery (2.00 to 4.00) — Pediatrics (2.00 to 4.30) — Gynecology Orthopaedics Psychiatry (Subgroups of Surgery Group) (4.00 to 5.00)	(Whole Class) Clinical Pathology — Laboratory *Bressler 5*	Surgery (2.00 to 4.00) — Pediatrics (2.00 to 4.30) — Gynecology Orthopaedics Psychiatry (Subgroups of Surgery Group) (4.00 to 5.00)	
4.00 to 5.00	(Whole Class) Hygiene and Public Health *C. H.*	(Whole Class) Physical Diagnosis, Psychiatry, Legal Medicine *C. H.*				

* First Semester. † Second Semester.

THIRD YEAR SCHEDULE

SCHEDULE 2

Hours	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
8.30 to 9.30	Same as Schedule 1					
9.30 to 10.20	Pediatrics *C. H.*	Medicine *C. H.*	Medicine *C. H.*	Therapeutics *C. H.*	Medicine *C. H.*	Neurology *C. H.*
10.30 to 12.30	Operative Surgery—*Bressler 6* Medical and Surgical Dispensaries—(Univ. and Mercy Sections)					
12.30 to 1.00	Lunch					
1.00 to 2.00	Same as Schedule 1		Medical Clinic *Amp.*	Same as Schedule 1	Psychiatry (9 weeks) Dermatology (6 weeks) N.B.—The whole section reports to psychiatry for first three weeks, then subdivides. *Bressler 2* for first 3 wks. U. H. Disp. Last 12 wks.	
2.00 to 4.00			Ophthalmoscopy (5 weeks) *B. E. H.* — Obstetrics (5 weeks) Univ. Hosp. Disp. — Otology (5 wks.) *Bressler 5 Conf. Room*			
4.00 to 5.00					Obstetrics *C. H.*	

The Junior Class will be divided into two sections—A and B. Each section reports to classes in keeping with the following schedule assignment, in which the letters represent the class sections and the numerals indicate the schedules to be followed for the 15-week periods shown.

Schedule Assignment

Periods	*Sections and Schedules*
September 25 to January 24	A-1, B-2
February 2 to May 16	B-1, A-2

Locations of Lecture Halls, etc.

A. H.—Anatomical Hall, Upper Hall, N. E. Cor. Lombard and Greene Streets.
Amp.—Wilson Memorial Amphitheatre, New University Hospital, Eighth Floor.
B. C. H.—Baltimore City Hosps., 4940 Eastern Ave.
B. E. H.—Baltimore Eye, Ear and Throat Hospital, 1214 Eutaw Place.
Bressler—Bressler Building, 29 S. Greene Street.
C. H.—Chemical Hall, Lower Hall, N. E. Cor. Lombard and Greene Streets.
Univ. Hosp.—New University Hospital, Greene and Redwood Streets.
U. H. Disp.—Old Hospital Building, S. W. Cor. Lombard and Greene Streets.
31—31 South Greene Street.
Clinical Pathology Laboratory—Fifth Floor, Bressler Building.
Pathology Laboratory—31 South Greene Street, Special Rooms, Basement.

Mid-Year Examinations—January 26–31, 1942
Final Examinations—May 18–30, 1942

FOURTH YEAR SCHEDULE

SEPTEMBER 25, 1941 TO MAY 30, 1942

Hours	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
9.00 to 11.00	Ward Classes — (a) Medicine (a) Surgery (b) Obstetrics (d) Pediatrics	Ward Classes — (a) Medicine (a) Surgery (b) Gynecology (c) Gynecology	Ward Classes — (a) Medicine (a) Surgery (b) Obstetrics (d) Pediatrics	Ward Classes — (a) Medicine (a) Surgery (b) Gynecology (c) Gynecology	Ward Classes — (a) Medicine (a) Surgery (b) Obstetrics (d) Pediatrics	Ward Classes — (a) Medicine (a) Surgery (b) Gynecology (d) Pediatrics
11.00 to 12.00	Orthopaedic Surgery *Univ. Sec. Amp.* *P. & S. Sec. 51*	Medical Clinic *Univ. Sec. Amp.* Surgical Pathology *P. & S. Sec. 40*	Clinical Pathological Conference *Univ. Sec. C. H.* *P. & S. Sec. 34*	Surgical Clinic *Univ. Sec. Amp.* *P. & S. Sec. 51*	Medical Clinic *Univ. Sec. Amp.* *P. & S. Sec. 34*	Pediatric Clinic *Univ. Sec. Amp.* *P. & S. Sec. 34*
12.00 to 2.00	Dispensary Lunch and Transfer	Dispensary and Lunch	Dispensary Lunch and Transfer	Dispensary and Lunch	Dispensary Lunch and Transfer	Dispensary
2.15 to 3.15	Dermatology Clinic *Univ. Sec.* { *Disp. Amp.* *P. & S. Sec. 34*	Neurology Clinic *Univ. Sec. Amp.* *P. & S. Sec. 34*	Eye and Ear Clinic (Full Class at Univ. Hosp.) *Amp.*	Obstetrical Clinic (Full Class at Univ. Hosp.) *Amp.*	Hygiene and Pulbic Health Sept. 26 to Oct. 31 Gastro-Enterology Clinic Nov. 7 (Full Class at Univ. Hosp.) *Amp.*	
3.30 to 5.00	*P. & S. Sect.* and *Univ. Sect.* Ward Classes	*P. & S. Sect.* Ward Classes	*P. & S. Sect.* Ward Classes	*P. & S. Sect.* Ward Classes	*P. & S. Sect.* Ward Classes	
		Medicine	Public Health	Medicine	Neurology	
		Orthopaedics	Nose and Throat	Proctology	Roentgenology	
		Pediatrics		Pediatrics	Psychiatry *Amp.*	
3.30 to 5.00	See special schedule Medical School bulletin board	*Univ. Sect.* Ward Classes	*Univ. Sect.* Ward Classes	*Univ. Sect.* Ward Classes	*Univ. Sect.* Ward Classes	
		Medicine	Public Health	Therapeutics	Neurology	
		Proctology	Urology *Amp.*	Nose and Throat	Orthopaedic Surgery (*Kernan Hospital*)	
		Oncology (3.30–4.30) *Amp.*	Eye and Ear		Psychiatry	

(a)—Univ. and P. & S. Sections.
(b)—Univ. Section.
(c)—Whole P. & S. special group.
(d)—P. & S. special group, divided attendance at Univ. and P. & S.

The Senior Class is divided into two sections, which report, one at Lombard and Greene Streets, the other at Calvert and Saratoga Streets, for one semester each, then rotate.

Each section of the class is divided into three groups—Medical, Surgical, and Special. These groups will rotate on the following dates:

First Semester		*Second Semester*	
1st period	Sept. 25–Nov. 1	1st period	Feb. 2–Mar. 7
2nd period	Nov. 3–Dec. 6	2nd period	Mar. 9–Apr. 11
3rd period	Dec. 8–Jan. 24	3rd period	Apr. 13–May 16

C. H.—Chemical Hall—N. E. Cor. Lombard and Greene Streets.
Amp.—Wilson Memorial Amphitheatre—New University Hospital.
Disp. Amp.—Old Hospital Building, fourth floor S. W. cor. Lombard and Greene Streets.
P. & S., 34—Second floor, Calvert and Saratoga Streets.
P. & S., 40, 51—Fourth floor, Calvert and Saratoga Streets.

Univ. Section-Dispensary schedule posted in Old Hospital Bldg.
P. & S. Section—obtain special schedule at Mercy Hospital

Mid-Year Examinations—January 26–31, 1942
Final Examinations—May 18–23, 1942

GRADUATES, JUNE 1, 1940

UNIVERSITY OF MARYLAND SCHOOL OF MEDICINE AND COLLEGE OF PHYSICIANS AND SURGEONS

Algire, Glenn Horner, B.S.......Maryland
Andrews, S. Ralph, Jr., A.B.....Maryland
Arney, William Charles, B.S. North Carolina
Baier, John Cletus.............Maryland
Bailey, Walter Levi, A.B.....Pennsylvania
Barker, Daniel Cleveland, A.B. Connecticut
Beacham, Edmund George, B.S. Maryland
Biehl, Harold Paul, A.B.........Maryland
Borden, Jesse Nachlas, A.B......Maryland
Brinsfield, Irving Carlton, A.B..Maryland
Caplan, Lester Harold, A.B.....Maryland
Chandler, Weldon Porter, B.S. North Carolina
Clifford, Robert Henry, Jr., B.S. New Jersey
Cole, John Totterdale...............Ohio
Correll, Paul Harvey, A.B.......Maryland
Daniel, Louie Samuel, B.S. North Carolina
Daue, Edwin Oliver, Jr., B.S....Maryland
De Luca, Joseph, B.S........Rhode Island
Dent, Charles Frederic......West Virginia
Don Diego, Leonard Vincent, B.S. New York
Duffy, William Carroll, A.B.....Maryland
Dwyer, James Richard, B.S..Pennsylvania
Freeman, James Albert, Jr...West Virginia
Fusting, William Hammond, B.S. Maryland
Gassaway, William Farrow.....Maryland
Gibbs, Robert Louis, A.B..North Carolina
Glick, Irving Van der Veere, A.B. New York
Graham, Walter Raleigh, A.B. North Carolina
Guzman-Lopez, Luis Roberto.Puerto Rico
Hecht, Morton, Jr., B.S.........Maryland
Henning, Emil Heller, Jr., A.B. Maryland
Heyman, Albert...............Maryland
Hooton, Elizabeth Louise, B.S.. Maryland
Hope, Daniel, Jr.............. Maryland
Igartua-Cardona, Susana.. Puerto Rico
Inloes, Benjamin Harrison, Jr....Maryland
Jamison, William Parks, B.S. West Virginia
Jorgensen, Louis Cecil, A.B..........Utah
Karns, James Roscoe...........Maryland
Kirchick, Julian Gilbert, A.B....New York
Kohn, Schuyler George, B.S.....Maryland
Krieg, Edward Louis Joseph....Maryland
Kurland, Albert Alexander......Maryland
Lartz, Robert Eshelman, B.S. Pennsylvania
Ling, William Soy Ming, A.B...New York
Livingood, William Cook, B.S. Pennsylvania
Loker, Frank Ford, B.S.........Maryland
Maccubbin, Harry Pearce, B.S..Maryland
Markline, Simeon Van Trump, A.B. Maryland
Martin, Clarence Wilbur........Maryland
Maryanov, Alfred Richard.....New York
Mathers, Daniel Hutchinson, A.B. Maryland
McCann, Harold Francis, A.B. West Virginia
McClung, James Edward, A.B. West Virginia
McClung, William Dennis...West Virginia
McDaniel, George Croxton, A.B. Maryland
McKinnon, William James, A.B. North Carolina
Meade, Forest Chauncey.......Maryland
Miceli, Joseph, A.B.............Maryland
Molz, Edward Louis, B.S.......Maryland
Murphy, Frederick Elbert, Jr., A.B. Georgia
Muse, William Travers, B.S.....Maryland
Myers, George Roger, Jr., A.B..Maryland
O'Hara, James Francis, B.S..........Ohio
Pico-Santiago, Guillermo......Puerto Rico
Pierpont, Ross Zimmerman.....Maryland
Pigford, Robert Toms.....North Carolina
Platt, William.................Maryland
Pollock, Arthur Edgar, B.S. .Pennsylvania
Posner, Leonard, B.S.... ..... New York
Pound, John Costello, A.B......Maryland
Rhode, Charles Martin, A.B....Maryland
Richter, Conrad Louis.........Maryland
Robinson, Raymond Clarence Vail Maryland
Roop, Donald James, A.B.......Maryland
Rothschild, Carl Eliot, B.S.........China
Russell, Thomas Edgie, Jr., B.S. Maryland
Russillo, Philip Joseph, A.B. . .New Jersey
Schlesinger, George Gerard, B.S. New York
Sloan, Joseph Wright, B.S.....New Jersey
Smith, James Brady, B.S........Maryland
Smith, Ruby Arden.........West Virginia
Squillante, Orlando John, B.S. Rhode Island
Stayton, Howard Nehemiah, Jr., A.B. Delaware
Supik, William Joseph..........Maryland
Tankin, Louis Haberer, A.B.....Maryland

Thompson, Alexander Frank, A.B. North Carolina
Tompakov, Samuel, A.B. . . . . .Maryland
Townshend, Wilfred Henry, Jr., A.B. Maryland
Trevor, William, A.B. . . . . .Maryland
Triplett, William Carryl. . . . .West Virginia
Waite, Merton Theodore. . . . . . . .Maryland
Weeks, William Earl, B.S. .North Carolina
Wilkins, Jesse Lee, B.S. . . . . .Maryland
Williams, Herman Joseph, B.S. Pennsylvania
Williams, Richard Talbot, A.B. Maryland
Wilson, Harry Thomas, Jr. . . . . . . .Maryland
Wolff, William Irwin, B.S. . . . . . .New York
Wright, James Rhodes, A.B. North Carolina
Zinkin, Solomon Bernard, A.B. New Jersey

HONORS

UNIVERSITY PRIZE GOLD MEDALS

JAMES ROSCOE KARNS
WILLIAM IRWIN WOLFF

CERTIFICATES OF HONOR

JOSEPH WRIGHT SLOAN
CARL ELIOT ROTHSCHILD
LESTER HAROLD CAPLAN
SAMUEL TOMPAKOV
WILLIAM SOY MING LING

The Dr. A. Bardley Gaither Memorial Prize of $25.00 for the best work in genito-urinary surgery during the senior year. . . . . .CHARLES MARTIN RHODE

INTERNSHIPS—GRADUATES OF 1940

July 1, 1940 to June 30, 1941

Algire, Glenn Horner. . . . . .University Hospital, Baltimore, Maryland
Andrews, Stephen Ralph, Jr. . . . . .Church Home & Infirmary, Baltimore, Maryland
Arney, William Charles. . . . . .Paterson General Hospital, Paterson, New Jersey
Baier, John Cletus. . . . . .St. Joseph's Hospital, Baltimore, Maryland
Bailey, Walter Levi. . . . . .York Hospital, York, Pennsylvania
Barker, Daniel Cleveland. . . . . .Waterbury Hospital, Waterbury, Connecticut
*★Beacham, Edmund George . . . . .Baltimore City Hospitals, Baltimore, Maryland
Biehl, Harold Paul. . . . . .Maryland General Hospital, Baltimore, Maryland
Borden, Jesse Nachlas. . . . . .Sinai Hospital, Baltimore, Maryland
Brinsfield, Irving Carlton. . . . . .Maryland General Hospital, Baltimore, Maryland
Caplan, Lester Harold. .University Hospital, Baltimore, Maryland
Chandler, Weldon Porter
North Carolina Baptist Hospital, Winston-Salem, North Carolina
Clifford, Robert Henry, Jr. . . . . .Union Memorial Hospital, Baltimore, Maryland
Cole, John Totterdale. . . . . .Church Home & Infirmary, Baltimore, Maryland
Correll, Paul Harvey. . . . . .Mercy Hospital, Baltimore, Maryland
Daniel, Louie Samuel. . . . . .City Hospital, Winston-Salem, North Carolina
Daue, Edwin Oliver, Jr. .University Hospital, Baltimore, Maryland
De Luca, Joseph. . . . . .St. Mary's Hospital, Waterbury, Connecticut
Dent, Charles Frederic. . . . . . .Western Pennsylvania Hospital, Pittsburgh, Pennsylvania
Don Diego, Leonard Vincent. . . . . .Coney Island Hospital, Brooklyn, New York
Duffy, William Carroll. . . . . .Mercy Hospital, Baltimore, Maryland
Dwyer, James Richard. . . . . .St. Agnes Hospital, Baltimore, Maryland

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*Did not complete the period.

Freeman, James Albert, Jr.	Mercy Hospital, Baltimore, Maryland
Fusting, William Hammond	Bon Secours Hospital, Baltimore, Maryland
Gassaway, William Farrow	Baltimore City Hospitals, Baltimore, Maryland
Gibbs, Robert Louis	University Hospital, Baltimore, Maryland
Glick, Irving Van der Veere	Harlem Hospital, New York City
Graham, Walter Raleigh	University Hospital, Baltimore, Maryland
Guzman-Lopez, Luis Roberto	University Hospital of the School of Tropical Medicine of the Columbia University, San Juan, Puerto Rico
Hecht, Morton, Jr.	Sinai Hospital, Baltimore, Maryland
Henning, Emil Heller, Jr.	Maryland General Hospital, Baltimore, Maryland
Heyman, Albert	Baltimore City Hospitals, Baltimore, Maryland
Hooton, Elizabeth Louise	Wilkes-Barre General Hospital, Wilkes-Barre, Pennsylvania
Hope, Daniel, Jr.	St. Agnes Hospital, Baltimore, Maryland
Igartua-Cardona, Susana	Bayamon District Charity Hospital, Bayamon, Puerto Rico
Inloes, Benjamin Harrison, Jr.	University Hospital, Baltimore, Maryland
*Jamison, William Parks	Western Pennsylvania Hospital, Pittsburgh, Pennsylvania
Jorgensen, Louis Cecil	Thomas D. Dee Hospital, Ogden, Utah
Karns, James Roscoe	University Hospital, Baltimore, Maryland
Kirchick, Julian Gilbert	St. Vincent's Hospital, Bridgeport, Connecticut
Kohn, Schuyler George	Metropolitan Hospital, Welfare Island, New York
Krieg, Edward Louis Joseph	Bon Secours Hospital, Baltimore, Maryland
Kurland, Albert Alexander	Sinai Hospital, Baltimore, Maryland
Lartz, Robert Eshelman	Pittsburgh Medical Center, Pittsburgh, Pennsylvania
Ling, William Soy Ming	Misericordia Hospital, New York City
Livingood, William Cook	Mercy Hospital, Baltimore, Maryland
Loker, Frank Ford	Mercy Hospital, Baltimore, Maryland
Maccubbin, Harry Pearce	University Hospital, Baltimore, Maryland
Markline, Simeon Van Trump	St. Vincent's Hospital, Jacksonville, Florida
*★Martin, Clarence Wilbur	St. Joseph's Hospital, Baltimore, Maryland
Maryanov, Alfred Richard	Knickerbocker Hospital, New York City
Mathers, Daniel Hutchinson	St. Vincent's Hospital, Jacksonville, Florida
McCann, Harold Francis	Mercy Hospital, Pittsburgh, Pennsylvania
**McClung, James Edward	Ohio Valley General Hospital, Wheeling, West Virginia
McClung, William Dennis	Mercy Hospital, Baltimore, Maryland
McDaniel, George Croxton	California Hospital, Los Angeles, California
McKinnon, William James	Maryland General Hospital, Baltimore, Maryland
Meade, Forest Chauncey	Mercy Hospital, Baltimore, Maryland
Miceli, Joseph	St. John's Hospital, Cleveland, Ohio
Molz, Edward Louis	St. Joseph's Hospital, Baltimore, Maryland
Murphy, Frederick Elbert, Jr.	Piedmont Hospital, Atlanta, Georgia
Muse, William Travers	St. Agnes Hospital, Baltimore, Maryland
Myers, George Roger, Jr.	Medical Corps, U. S. Navy
O'Hara, James Francis, Jr.	Mercy Hospital, Canton, Ohio
Pico-Santiago, Guillermo	Presbyterian Hospital, Santurce, Puerto Rico
Pierpont, Ross Zimmerman	Maryland General Hospital, Baltimore, Maryland
Pigford, Robert Toms	Union Memorial Hospital, Baltimore, Maryland
Platt, William	St. Joseph's Hospital, Lexington, Kentucky
Pollock, Arthur Edgar	Mercy Hospital, Altoona, Pennsylvania

**Did not complete the period; was released to another hospital.

Posner, Leonard	Coney Island Hospital, Brooklyn, New York
Pound, John Costello	St. Agnes Hospital, Baltimore, Maryland
Rhode, Charles Martin	University Hospital, Baltimore, Maryland
Richter, Conrad Louis	St. Joseph's Hospital, Baltimore, Maryland
Robinson, Raymond Clarence Vail	Maryland General Hospital, Baltimore, Maryland
Roop, Donald James	Mercy Hospital, Baltimore, Maryland
Rothschild, Carl Eliot	Mount Sinai Hospital, Cleveland, Ohio
Russell, Thomas Edgie, Jr.	Bon Secours Hospital, Baltimore, Maryland
Russillo, Philip Joseph	Misericordia Hospital, New York City
Schlesinger, George Gerard	Morrisania City Hospital, New York City
Sloan, Joseph Wright	Kings County Hospital, Brooklyn, New York
Smith, James Brady	St. Agnes Hospital, Baltimore, Maryland
Smith, Ruby Arden	Charleston General Hospital, Charleston, West Virginia
Squillante, Orlando John	Danbury Hospital, Danbury, Connecticut
Stayton, Howard Nehemiah, Jr.	Delaware Hospital, Wilmington, Delaware
Supik, William Joseph	St. Joseph's Hospital, Baltimore, Maryland
Tankin, Louis Haberer	Sinai Hospital, Baltimore, Maryland
Thompson, Alexander Frank	University Hospital, Baltimore, Maryland
Tompakov, Samuel Victor	Sinai Hospital, Baltimore, Maryland
Townshend, Wilfred Henry, Jr.	University Hospital, Baltimore, Maryland
Trevor, William	Bryn Mawr Hospital, Bryn Mawr Pennsylvania
Triplett, William Carryl	Ohio Valley General Hospital, Wheeling, West Virginia
Waite, Merton Theodore	Sibley Memorial Hospital, Washington, District of Columbia
Weeks, William Earl	Mercy Hospital, Baltimore, Maryland
Wilkins, Jesse Lee	Doctors Hospital, Washington, District of Columbia
Williams, Herman Joseph	U. S. Marine Hospital, Baltimore, Maryland
Williams, Richard Talbot	University Hospital, Baltimore, Maryland
*★Wilson, Harry Thomas, Jr.	St. Joseph's Hospital, Baltimore, Maryland
Wolff, William Irwin	Bellevue Hospital (Second Cornell Medical Division), New York City
Wright, James Rhodes	Maryland General Hospital, Baltimore, Maryland
Zinkin, Solomon Bernard	Morrisania City Hospital, New York City

GRADUATES, JUNE 7, 1941

UNIVERSITY OF MARYLAND SCHOOL OF MEDICINE AND COLLEGE OF PHYSICIANS AND SURGEONS

Alberti, Aurora Frances, A.B.	New York
Alexander, Fred., A.B.	New Jersey
Arnett, Jerome Cayton, B.S.	West Virginia
Barnett, Charles Phelps, A.B.	Maryland
Baxley, Joshua Warfield, III, B.S.	Maryland
Bowen, Joseph John, B.S.	Connecticut
Brooks, Julius Culpepper, Jr., B.S.	Tennessee
Bundick, William Ross	Maryland
Carter, John Marshall, A.B., B.S.	West Virginia
Checket, Pierson Melvin, A.B.	Maryland
Chiqués, Carlos Miguel	Puerto Rico
Cloninger, Charles Edgar, A.B.	North Carolina
Conlen, Richard Alexis, B.S.	New Jersey
Cooper, LeRoy Gerald, A.B.	Pennsylvania
Crecca, Joseph Vincent, B.S.	New Jersey
Croce, Gene Albert, B.S.	Rhode Island
Cruikshank, Dwight Phelph, Jr., A.B.	West Virginia
Culler, John McCleary, A.B.	Maryland
DeVincentis, Michael Louis, B.S.	Maryland
Diez-Gutierrez, Emilio	Puerto Rico
DiPaula, Anthony Francis, A.B.	Maryland
Esnard, John Edward	California
Evola, Camille Mary, A.B.	New York
Frey, Edward Leonard, Jr., A.B.	Maryland
Garcia-Blanco, José	Puerto Rico
Gelber, Julius, A.B.	New York

Goodman, William, A.B........Maryland
Graziano, Theodore Joseph, A.B. Maryland
Hedrick, Thomas Ardis.....West Virginia
Hershner, Newton Webster, Jr., B.S. Pennsylvania
Hollander, Asher, A.B..........Maryland
Huffman, Pearl Trogdon, A.B. North Carolina
Hunter, James Stanley, Jr......Maryland
Jaffe, Vita Rebecca, B.S........New York
Kemp, Norval Foard, A.B......Maryland
Krulevitz, Keaciel Kenneth, B.S. Maryland
Lach, Frank Edward, B.S.....New Jersey
Leslie, Franklin Earl, A.B.......Maryland
Levinson, Lorman Leon, A.B....Maryland
Licha, José Salomon..........Puerto Rico
Lowe, William Cook, B.S.......Maryland
Lusby, Thomas Frank, A.B.....Maryland
Malouf, Raymond N., B.S...........Utah
Mandel, Jacob Barry, M.S....New Jersey
Mitchell, William Arthur.......Maryland
Molinari, José Gilberto.......Puerto Rico
Morgan, Margaret...............Indiana
Morris, Felix Raymond, B.S...Connecticut
Morrison, William Herbert.......Maryland
Nolan, James Joseph, B.S......Maryland
Novoa-Caballero, Miguel.....Puerto Rico
Ortiz, Idalia Ortiz, B.S........Puerto Rico
Palmer, Margaret Virginia, M.A. Maryland
Pasamanick, Benjamin, A.B....New York
Pearcy, Thompson, A.B.....West Virginia
Perman, Joshua Melvin, A.B....Maryland
Phrydas, Irene, A.B.......North Carolina
Pruitt, Charles Eugene, A.B....Maryland
Renna, Francis Stanley, A.B...New Jersey
Revell, Walter Jones, B.S.........Georgia
Richardson, Charles, Jr.........Maryland
Richmond, Marion Ballard, B.S. Maryland
Richter, Christian Frederick, Jr., B. S. Maryland
Rosenberg, Jonas Samuel, A.B..New York
Rossberg, Clyde Arthur, A.B....Maryland
Sasscer, Robert Bowie, A.B.....Maryland
Sawyer, William Hamilton, A.B. North Carolina
Scholl, John Andrew, A.B., B.S. West Virginia
Schwartz, Stanley Eugene, B.S...New York
Seigman, Edwin Lincoln, Jr., A.B. Maryland
Shannon, Edward Patrick, Jr., B.S. New York
Sheehan, Joseph Chester, B.S...Maryland
Sherrill, Elizabeth Brown, B.S...Maryland
Sims, Thomas Courtland, A.B., B.S. West Virginia
Skitarelic, Benedict, A.B., B.S. West Virginia
Spencer, Tracy Neil, Jr., A.B. North Carolina
Spinnler, Henry Robert, B.S...New Jersey
Stevens, John Sutehall, A.B...Connecticut
Strayer, Webster Mills, Jr., A.B..Maryland
Thompson, Raymond Kief, B.S..Maryland
Trevaskis, Richard White, A.B..Maryland
Ulrich, George John, A.B.......Maryland
Virusky, Edmund Joseph, B.S. Pennsylvania
Walker, James Haward, A.B. West Virginia
Wall, Lester Aubrey, Jr., A.B...Maryland
Watkins, Dayton O'Lander, B.S. Maryland
Wells, John Bernard, Jr., B.S....Maryland
Wilder, Thomas Carroll......Minnesota
Wilson, Edwin Forrest, A.B...New York
Yanagisawa, Kazuo, A.B........California
Young, John David, Jr., A.B....Maryland
Zierler, Kenneth Levie, A.B.....Maryland

HONORS

University Prize Gold Medal..........................JAMES STANLEY HUNTER, JR.

CERTIFICATES OF HONOR

MARGARET VIRGINIA PALMER
NORVAL FOARD KEMP
JOSÉ SALOMON LICHA
GEORGE JOHN ULRICH
LESTER AUBREY WALL, JR.
RAYMOND KIEF THOMPSON

The Dr. A. Bradley Gaither Memorial Prize of $25.00 for the best work in genito-urinary surgery during the senior year..........................RAYMOND N. MALOUF

INTERNSHIPS—GRADUATES OF 1941

Effective July 1, 1941

Alberti, Aurora Frances	Jersey City Medical Center, Jersey City, New Jersey
Alexander, Fred	St. Francis Hospital, Pittsburgh, Pennsylvania
Arnett, Jerome Cayton	Lutheran Hospital, Cleveland, Ohio
Barnett, Charles Phelps	Mercy Hospital, Baltimore, Maryland
Baxley, Joshua Warfield, III	University Hospital, Baltimore, Maryland
Bowen, Joseph John	St. Mary's Hospital, Waterbury, Connecticut
Brooks, Julius Culpepper, Jr.	The Baroness Erlanger Hospital, Chattanooga, Tennessee
Bundick, William Ross	The Baroness Erlanger Hospital, Chattanooga, Tennessee
Carter, John Marshall	Mercy Hospital, Pittsburgh, Pennsylvania
Checket, Pierson Melvin	West Baltimore General Hospital, Baltimore, Maryland
Chiqués, Carlos Miguel	Bayamon District Charity Hospital, Bayamon, Puerto Rico
Cloninger, Charles Edgar	Garfield Memorial Hospital, Washington, District of Columbia
Conlen, Richard Alexis	Cooper Hospital, Camden, New Jersey
Cooper, LeRoy Gerald	York Hospital, York, Pennsylvania
Crecca, Joseph Vincent	Newark City Hospital, Newark, New Jersey
Croce, Gene Albert	St. Mary's Hospital, Waterbury, Connecticut
Cruikshank, Dwight Phelph, Jr.	Union Memorial Hospital, Baltimore, Maryland
Culler, John McCleary	Church Home and Infirmary, Baltimore, Maryland
DeVincentis, Michael Louis	Mercy Hospital, Baltimore, Maryland
Diez-Gutierrez, Emilio	Misericordia Hospital, Philadelphia, Pennsylvania
DiPaula, Anthony Francis	Hospital for the Women of Maryland, Baltimore, Maryland
Esnard, John Edward	Los Angeles County General Hospital, Los Angeles, California
Evola, Camille Mary	North Hudson Hospital, Weehauken, New Jersey
Frey, Edward Leonard, Jr.	Mercy Hospital, Baltimore, Maryland
Garcia-Blanco, José	James M. Jackson Memorial Hospital, Miami, Florida
Gelber, Julius	Sinai Hospital, Baltimore, Maryland
Goodman, William	Baltimore City Hospitals, Baltimore, Maryland
Graziano, Theodore Joseph	Union Memorial Hospital, Baltimore, Maryland
Hedrick, Thomas Ardis	Mercy Hospital, Baltimore, Maryland
Hershner, Newton Webster, Jr.	Harrisburg Hospital, Harrisburg, Pennsylvania
Hollander, Asher	Mount Sinai Hospital, Cleveland, Ohio
Huffman, Pearl Trogdon	Baltimore City Hospitals, Baltimore, Maryland
Hunter, James Stanley, Jr.	University Hospital, Baltimore, Maryland
Jaffe, Vita Rebecca	Israel Zion Hospital, Brooklyn, New York
Kemp, Norval Foard	University Hospital, Baltimore, Maryland
Krulevitz, Keaciel Kenneth	Sinai Hospital, Baltimore, Maryland
Lach, Frank Edward	Grace Hospital, New Haven, Connecticut
Leslie, Franklin Earl	Union Memorial Hospital, Baltimore, Maryland
Levinson, Lorman Leon	University Hospital, Baltimore, Maryland
Licha, José Salomon	Knickerbocker Hospital, New York, New York
Lowe, William Cook	Mercy Hospital, Baltimore, Maryland
Lusby, Thomas Frank	Maryland General Hospital, Baltimore, Maryland
Malouf, Raymond N.	Salt Lake County General Hospital, Salt Lake City, Utah
Mandel, Jacob Barry	Jersey City Medical Center, Jersey City, New Jersey
Mitchell, William Arthur	Maryland General Hospital, Baltimore, Maryland
Molinari, José Gilberto	Bayamon District Charity Hospital, Bayamon, Puerto Rico
Morgan, Margaret	Grasslands Hospital, Valhalla, New York
Morris, Felix Raymond	St. Vincent's Hospital, Bridgeport, Connecticut

Morrison, William Herbert	York Hospital, York, Pennsylvania
Nolan, James Joseph	Union Memorial Hospital, Baltimore, Maryland
Novoa-Caballero, Miguel	Presbyterian Hospital, San Juan, Puerto Rico
Ortiz, Idalia Ortiz	Presbyterian Hospital, Santurce, Puerto Rico
Palmer, Margaret Virginia	University Hospital, Baltimore, Maryland
Pasamanick, Benjamin	Harlem Hospital, New York, New York
Pearcy, Thompson	Union Memorial Hospital, Baltimore, Maryland
Perman, Joshua Melvin	West Baltimore General Hospital, Baltimore, Maryland
Phrydas, Irene	Metropolitan Hospital, Welfare Island, New York
Pruitt, Charles Eugene	Church Home and Infirmary, Baltimore, Maryland
Renna, Francis Stanley	Elizabeth General Hospital, Elizabeth, New Jersey
Revell, Walter Jones	University Hospital, Baltimore, Maryland
Richardson, Charles, Jr.	Central Dispensary and Emergency Hospital, Washington, District of Columbia
Richmond, Marion Ballard	Henry Ford Hospital, Detroit, Michigan
Richter, Christian Frederick, Jr.	Bon Secours Hospital, Baltimore, Maryland
Rosenberg, Jonas Samuel	Beth Israel Hospital, New York, New York
Rossberg, Clyde Arthur	St. Agnes Hospital, Baltimore, Maryland
Sasscer, Robert Bowie	Mercy Hospital, Baltimore, Maryland
Sawyer, William Hamilton	St. Agnes Hospital, Baltimore, Maryland
Scholl, John Andrew	Mercy Hospital, Pittsburgh, Pennsylvania
Schwartz, Stanley Eugene	Queens General Hospital, Jamaica, Long Island, New York
Seigman, Edwin Lincoln, Jr.	University Hospital, Baltimore, Maryland
Shannon, Edward Patrick, Jr.	Letterman General Hospital, San Francisco, California
Sheehan, Joseph Chester	Mercy Hospital, Baltimore, Maryland
Sherrill, Elizabeth Brown	Hospital for the Women of Maryland, Baltimore, Maryland
Sims, Thomas Courtland	Charleston General Hospital, Charleston, West Virginia
Skitarelic, Benedict	Mercy Hospital, Pittsburgh, Pennsylvania
Spencer, Tracy Neil, Jr.	Mercy Hospital, Baltimore, Maryland
Spinnler, Henry Robert	St. Joseph's Hospital, Patterson, New Jersey
Stevens, John Sutehall	Mercy Hospital, Baltimore, Maryland
Strayer, Webster Mills, Jr.	University Hospital, Baltimore, Maryland
Thompson, Raymond Kief	University Hospital, Baltimore, Maryland
Trevaskis, Richard White, Jr.	Chester County Hospital, West Chester, Pennsylvania
Ulrich, George John	University Hospital, Baltimore, Maryland
Virusky, Edmund Joseph	St. Francis Hospital, Pittsburgh, Pennsylvania
Walker, James Haward	University Hospital, Baltimore, Maryland
Wall, Lester Aubrey, Jr.	Grasslands Hospital, Valhalla, New York
Watkins, Dayton O'Lander	Gallinger Municipal Hospital, Washington, District of Columbia
Wells, John Bernard, Jr.	St. Vincent's Hospital, New York, New York
Wilder, Thomas Carroll	University Hospital, Baltimore, Maryland
Wilson, Edwin Forrest	Metropolitan Hospital, Welfare Island, New York
Yanagisawa, Kazuo	St. Clare's Hospital, New York, New York
Young, John David, Jr.	Maryland General Hospital, Baltimore, Maryland
Zierler, Kenneth Levie	Sinai Hospital, Baltimore, Maryland

MATRICULATES

FOURTH YEAR CLASS 1940–1941

Alberti, Aurora Frances, *A.B., Smith College, 1937*	New York
Alexander, Fred, *A.B., St. John's College, 1937*	New Jersey
Arnett, Jerome Cayton, *B.S., West Virginia Wesleyan College, 1935; B.S., West Virginia University School of Medicine, 1937–1939*	West Virginia
Barnett, Charles Phelps, *A.B., The Johns Hopkins University, 1937*	Maryland
Baxley, Joshua Warfield, III, *B.S., University of Maryland, 1938*	Maryland
Bowen, Joseph John, *B.S., University of Maryland, 1938*	Connecticut
Brooks, Julius Culpepper, Jr., *B.S., University of Chattanooga, 1937*	Tennessee
Bundick, William Ross, *University of Maryland*	Maryland
Carter, John Marshall, *A.B., West Virginia University, 1938; B.S., West Virginia University School of Medicine, 1937–1939*	West Virginia
Checket, Pierson Melvin, *A.B., The Johns Hopkins University, 1937*	Maryland
Chiques, Carlos Miguel, *University of Puerto Rico*	Puerto Rico
Cloninger, Charles Edgar, *A.B., Lenoir Rhyne College, 1937; University of North Carolina School of Medicine, 1937–1939*	North Carolina
Conlen, Richard Alexis, *B.S., College of William and Mary, 1935; University of North Carolina School of Medicine, 1937–1938*	New Jersey
Cooper, LeRoy Gerald, *A.B., The Johns Hopkins University, 1937*	Pennsylvania
Crecea, Joseph Vincent, *B.S., University of Maryland, 1935*	New Jersey
Croce, Gene Albert, *B.S., Rhode Island State College, 1937*	Rhode Island
Cruikshank, Dwight Phelph, Jr., *A.B., West Virginia University, 1937*	West Virginia
Culler, John McCleary, *A.B., Western Maryland College, 1937*	Maryland
DeVincentis, Michael Louis, *B.S., Loyola College, 1937*	Maryland
Diez-Gutierrez, Emilio, *University of Puerto Rico*	Puerto Rico
DiPaula, Anthony Francis, *A.B., The Johns Hopkins University, 1937*	Maryland
Esnard, John Edward, *Loyola University*	California
Evola, Camille Mary, *A.B., Hunter College, 1937*	New York
Frey, Edward Leonard, Jr., *A.B., The Johns Hopkins University, 1937*	Maryland
Garcia-Blanco, José, *University of Puerto Rico*	Puerto Rico
Gelber, Julius, *A.B., The Johns Hopkins University, 1936*	New York
Goodman, William, *A.B., The Johns Hopkins University, 1937*	Maryland
Graziano, Theodore Joseph, *A.B., The Johns Hopkins University, 1937*	Maryland
Hedrick, Thomas Ardis, *Marshall College*	West Virginia
Hershner, Newton Webster, Jr., *B.S., Dickinson College, 1937*	Pennsylvania
Hollander, Asher, *A.B., St. John's College, 1937*	Maryland
Huffman, Pearl Trogdon, *A.B., Meredith College, 1937; University of North Carolina School of Medicine, 1937–1939*	North Carolina
Hunter, James Stanley, Jr., *McGill University*	Maryland
Jaffe, Vita Rebecca, *B.S., University of Maryland, 1937*	New York
Kemp, Norval Foard, *A.B., St. John's College, 1937*	Maryland
Krulevitz, Keaciel Kenneth, *B.S., University of Maryland, 1937*	Maryland
Lach, Frank Edward, *B.S., The College of the City of New York, 1936*	New Jersey
Leslie, Franklin Earl, *A.B., St. John's College, 1937*	Maryland
Levinson, Lorman Leon, *A.B., The Johns Hopkins University, 1937*	Maryland
Licha, José Salomon, *Villanova College*	Puerto Rico
Lowe, William Cook, *B.S., University of Maryland, 1938*	Maryland
Lusby, Thomas Frank, *A.B., St. John's College, 1937*	Maryland
Malouf, Raymond N., *B.S., Utah State Agricultural College, 1937; University of Utah School of Medicine, 1937–1939*	Utah
Mandel, Jacob Barry, *B.S., M.S., University of Maryland, 1935, 1937*	New Jersey
Mitchell, William Arthur, *University of Maryland*	Maryland
Molinari, José Gilberto, *University of Maryland*	Puerto Rico
Morgan, Margaret, *University of Louisville; University of Louisville School of Medicine, 1937–1939*	Indiana
Morris, Felix Raymond, *B.S., University of Maryland, 1938*	Connecticut
Morrison, William Herbert, *The Johns Hopkins University*	Maryland
Nolan, James Joseph, *B.S., Loyola College, 1937*	Maryland
Novoa-Caballero, Miguel, *University of Puerto Rico*	Puerto Rico
Ortiz, Idalia Ortiz, *B.S., University of Puerto Rico, 1937*	Puerto Rico
Palmer, Margaret Virginia, *A.B., Goucher College, 1933, M.A., Columbia University, 1936*	Maryland
Pasamanick, Benjamin, *A.B., Cornell University, 1936*	New York

Pearcy, Thompson, *A.B., West Virginia University, 1938*................West Virginia
Perman, Joshua Melvin, *A.B., The Johns Hopkins University, 1937*.........Maryland
Phrydas, Irene, *A.B., Woman's College of the University of North Caroline, 1937; University of North Carolina School of Medicine, 1937–1939*.........North Carolina
Pruitt, Charles Eugene, *A.B., Duke University, 1937*.....................Maryland
Renna, Francis Stanley, *A.B., New York University, 1937*................New Jersey
Revell, Walter Jones, *B.S., University of Georgia, 1937*.....................Georgia
Richardson, Charles, Jr., *The Johns Hopkins University*..................Maryland
Richmond, Marion Ballard, *B.S., University of Maryland, 1937*............Maryland
Richter, Christian Frederick, Jr., *B.S., University of Maryland, 1937*.....Maryland
Rosenberg, Jonas Samuel, *A.B., Columbia University, 1937*................New York
Rossberg, Clyde Arthur, *A.B., The Johns Hopkins University, 1937*........Maryland
Sasscer, Robert Bowie, *A.B., St. John's College, 1937*.....................Maryland
Sawyer, William Hamilton, *A.B., University of North Carolina, 1937*...North Carolina
Scholl, John Andrew, *A.B., West Virginia University, 1937; B.S., West Virginia University School of Medicine, 1937–1939*........................West Virginia
Schwartz, Stanley Eugene, *B.S., University of Maryland, 1937*............New York
Seigman, Edwin Lincoln, Jr., *A.B., The Johns Hopkins University, 1937*....Maryland
Shannon, Edward Patrick, Jr., *B.S., Holy Cross College, 1937*............New York
Sheehan, Joseph Chester, *B.S., Georgetown University, 1937*................Maryland
Sherrill, Elizabeth Brown, *B.S., University of Maryland, 1939*............Maryland
Sims, Thomas Courtland, *A.B., West Virginia University, 1936; B.S., West Virginia University School of Medicine, 1935–1937*..................West Virginia
Skitarelic, Benedict, *A.B., West Virginia University, 1937; B.S., West Virginia University School of Medicine, 1937–1939*........................West Virginia
Spencer, Tracy Neil, Jr., *A.B., University of North Carolina, 1937*.....North Carolina
Spinnler, Henry Robert, *B.S., University of Virginia, 1937*..............New Jersey
Stevens, John Sutehall, *A.B., The Johns Hopkins University, 1937*.......Connecticut
Strayer, Webster Mills, Jr., *A.B., Western Maryland, 1936*...............Maryland
Thompson, Raymond Kief, *B.S., University of Maryland, 1937*.............Maryland
Trevaskis, Richard White, *A.B., Harvard College, 1937*..................Maryland
Ulrich, George John, *A.B., The Johns Hopkins University, 1933*...........Maryland
Virusky, Edmund Joseph, *B.S., Villanova College, 1937*.................Pennsylvania
Walker, James Haward, *A.B., West Virginia Wesleyan College, 1937*....West Virginia
Wall, Lester Aubrey, Jr., *A.B., St. John's College, 1937*..................Maryland
Watkins, Dayton O'Lander, *B.S., University of Maryland, 1937*...........Maryland
Wells, John Bernard, Jr., *B.S., Loyola College, 1937*......................Maryland
Wilder, Thomas Carroll, *University of Minnesota*.........................Minnesota
Wilson, Edwin Forrest, *A.B., Columbia University, 1937*..................New York
Yanagisawa, Kazuo, *A.B., University of California, 1935*..................California
Young, John David, Jr., *A.B., Bridgewater College, 1938*...................Maryland
Zierler, Kenneth Levie, *A.B., The Johns Hopkins University, 1936*.........Maryland

THIRD YEAR CLASS 1940–1941

Ahroon, William Alstrom, *A.B., The Johns Hopkins University, 1938*.......Maryland
Bacharach, David Nathan, Jr., *A.B., St. John's College, 1938*..............Maryland
Baldwin, Earl Rudolph, Jr., *B.S., Wake Forest College, 1939; Wake Forest College School of Medical Sciences, 1938–1940*........................North Carolina
Barthel, Robert Amthor, Jr., *B.S., University of Maryland, 1939*..........Maryland
Bassan, Morton Edward, *A.B., The Johns Hopkins University, 1938*........Maryland
Bennett, Van Boring, *A.B., University of North Carolina, 1937*........North Carolina
Bird, Joseph Gordon, *B.S., Florida Southern College, 1937*..................Maryland
Bowen, Francis Dorsey Thomas, *B.S., Villanova College, 1938*.............Maryland
Brodsky, Alexander Emmanuil, *B.S., University of Maryland, 1938*.........Maryland
Byerly, William Luther, Jr., *B.S., University of South Carolina, 1938*...South Carolina
Carey, Richard Alexius, *B.S., Loyola College, 1938*.......................Maryland
Coffman, Harry Franklin, II, *A.B., West Virginia University, 1938*.....West Virginia
Concilus, Frank, *A.B., West Virginia University, 1938*..................Pennsylvania
Corbett, Sybil, *B.S., Florida State College for Women, 1932; University of North Carolina School of Medicine, 1938–1940*...................................Florida
Cox, Matthew Mordica, *The Johns Hopkins University*...................Maryland
Crane, Warren Eugene, *University of Maryland*.......................New Jersey

Currence, William Ward, *B.S. in Chemistry, Virginia Military Institute, 1935; B.S., West Virginia University School of Medicine, 1938–1940*.......West Virginia
Davies, Thomas Eugene, *University of Maryland*......................Pennsylvania
Davila-Lopez, José G., *B.S., University of Puerto Rico, 1938*..............Puerto Rico
Davis, John Russell, Jr., *A.B., West Virginia University, 1936*..........West Virginia
Day, Newland Edward, *The Johns Hopkins University*....................Maryland
Dillinger, Karl Anthony, *West Virginia Wesleyan College*.............West Virginia
Dixon, Philip Lafayette, Jr., *B.S., Wake Forest College, 1939; Wake Forest College School of Medicine, 1938–1940*.............................North Carolina
Fallin, Herbert Kirk, *A.B., Western Maryland College, 1938*...............Maryland
File, Richard Cushing, *B.S., Washington and Jefferson College, 1938*...........Illinois
Franz, John Howard, *A.B., Randolph-Macon College, 1938*.................Maryland
Friedman, Marion, *B.S., University of Maryland, 1938*.....................Maryland
Fuertes, José Rodriguez, *B.S., University of Puerto Rico, 1938*...........Puerto Rico
Furnari, Joseph Charles, *B.S., University of Pittsburgh, 1938*...........Pennsylvania
Goldsmith, Jewett, *A.B., The Johns Hopkins University, 1938*..............Maryland
Gramse, Arthur Edward, *A.B., Yale University, 1937*................Massachusetts
Gregory, Exie Mildred, *West Virginia University*....................West Virginia
Hamburger, Morton Leonard, *B.S., University of Maryland, 1938*..........Maryland
Hubbard, Prevost, Jr., *B.S., Springfield College, 1938*.....................New York
Ingram, Albert Lester, Jr., *B.S., Davidson College, 1938*..................Delaware
Irwin, Robert Clark, *B.S., University of Maryland, 1939*.................New Jersey
Johnson, Hansford Fred, *B.S., Wake Forest College, 1938; Wake Forest College School of Medicine, 1938–1940*...Georgia
Jones, Everett Davis, *A.B., Western Maryland College, 1938*...............Maryland
Kardash, Theodore, *B.S., University of Maryland, 1938*....................Maryland
Keeley, Joseph Francis, Jr., *A.B., Harvard College, 1938*................Connecticut
Kenyon, Harold Arthur, *A.B., St. John's College, 1938*...............Massachusetts
Kiefer, Robert Allan, *A.B., Western Maryland College, 1937*..............Maryland
Klijanowicz, Stanley Benedict, *Ph.B., Loyola College, 1938*...............Maryland
Koleshko, Lawrence Jacob, *B.S., Washington College, 1938*.............Connecticut
Krepp, Martin William, Jr., *University of Maryland*.......................Maryland
Kroll, John Gregory, *A.B., Bethany College, 1938*.....................Pennsylvania
Kundahl, Paul Charles, *B.S., University of Maryland, 1940*...............Maryland
Link, Etta Carolyn, *B.S., University of Maryland, 1939*...................Maryland
Longwell, Robert Hamilton, *B.S., Juniata College, 1938*...............Pennsylvania
Lowitz, Irving Robert, *B.S., University of Maryland, 1938*.................Maryland
Lyons, Mary Louise, *A.B., West Virginia University, 1938; West Virginia University School of Medicine, 1938–1939*.......................................Ohio
Manganiello, Louis Otto, *A.B., Harvard College, 1937*..................Connecticut
Marino, Frank Sebastian, *A.B., Wesleyan College, 1938*................Connecticut
Matthews, Henry Steadman, *A.B., University of North Carolina, 1937* District of Columbia
Mazer, Robert, *B.S., University of Maryland, 1938*........................Maryland
McCosh, James Nathaniel, Jr., *B.A., The Johns Hopkins University, 1938*..Maryland
McGoogan, Malcolm Thomas, Jr., *B.S., University of Maryland, 1940*........Georgia
Meli, John James, *Waynesburg College*..............................Pennsylvania
Miller, Edgar Allen, Jr., *A.B., Gettysburg College, 1938*...............Pennsylvania
Moses, Robert Abram, *A.B., The Johns Hopkins University, 1938*...........Maryland
Mullins, George Roy, Jr., *West Virginia University*..................West Virginia
Orofino, Caesar Francis, *B.S., University of Maryland, 1939*.............New York
Osborne, John Carlton, *B.S., Loyola College, 1937*........................Maryland
Phelan, Patrick Carey, Jr., *A.B., Loyola College, 1935*....................Maryland
Phillips, Otto Charles, *A.B., The Johns Hopkins University, 1938*..........Maryland
Posey, Dale Morton, *B.S., Muhlenberg College, 1937*..................Pennsylvania
Raby, William Thomas, *B.S., The Citadel, 1938; University of North Carolina School of Medicine, 1938–1940*..............................North Carolina
*Rath, Maurice Monroe, *A.B., Indiana University, 1936*.................New Jersey
Ritchings, Edward Peyton, *A.B., St. John's College, 1938*.................Maryland
Rosin, John David, *The Johns Hopkins University, 1938*...................Maryland
Rousos, Anthony Peter, *A.B., Cornell University, 1938*..................New York
Sadler, Henry Harrison, Jr., *A.B., St. John's College, 1938*...............Maryland

*Did not complete the year.

Sadowsky, Wallace Hyman, *B.S., University of Maryland, 1939*............Maryland
Sborofsky, Isadore, *University of Maryland, Johns Hopkins University*....Maryland
Scott, Joseph Whiddon, *A.B., Duke University, 1938*........................Florida
Senter, William Jeffress, *B.S., Wake Forest College, 1938; Wake Forest College School of Medicine, 1938–1940*..................................North Carolina
Shipley, Edgar Roderick, *A.B., The Johns Hopkins University, 1938*........Maryland
Shub, Maurice Isaac, *A.B., The Johns Hopkins University, 1938*............Maryland
Shuman, Louis Harry, *A.B., St. John's College, 1938*......................Maryland
Stegmaier, James George, *B.S., University of Maryland, 1939*..............Maryland
Summa, Andrew Anthony James, *A.B., Syracuse University, 1938*...........New York
Townsend, Francis James, *A.B., St. John's College, 1938*..................Maryland
Traynor, Francis Willoughby, *B.S., University of Notre Dame, 1937*.......Maryland
Wallace, Joseph Jr., *A.B., University of Pennsylvania, 1938*.............Pennsylvania
Ward, Charles Monroe, *B.S., West Virginia Wesleyan College, 1937*.....West Virginia
Williams, Charles Herman, *A.B., Western Maryland College, 1937*..........Maryland
Williamson, Edgar Percival, Jr., *A.B., The Transylvania University, 1938*..Maryland
Zepp, Edwin Andrew, *A.B., West Virginia University, 1939; B.S., West Virginia University School of Medicine, 1938–1940*........................West Virginia
Zimmerman, Loy Miller, *B.S., University of Maryland, 1939*................Maryland

SECOND YEAR CLASS 1940–1941

Adam, Alberto Lotfalla, *Villanova College*............................Puerto Rico
Allsopp, Richard Charles, *University of Pittsburgh*..................Pennsylvania
Almodóvar, Ramón Ignacio, *West Virginia University*....................Puerto Rico
Baker, Emory Forester, *A.B., Stanford University, 1939*................Washington
Barnes, John David, *A.B., Harvard College, 1937*.....................Massachusetts
Bowen, Charles Vernon, Jr., *B.S., University of Maryland, 1939*...........Maryland
Brennan, Thomas Joseph, *B.S., Loyola College, 1939*.......................Maryland
Brooks, Ralph King, *A.B., West Virginia University, 1939*.............West Virginia
Brooks, Ross Chilton, *B.S., Loyola College, 1939*.........................Maryland
Bryson, William James, *A.B., Western Maryland College, 1939*..............Maryland
Chenowith, Ralph Stallings, *University of Maryland School of Pharmacy, Loyola College*...Maryland
Cohen, Harry, *B.S., University of Maryland, 1939*........................Maryland
Coughlin, John Benedict, *B.S., Niagara University, 1939*..............Pennsylvania
Courtney, Donald Lawrence, *University of Oregon*..........................Oregon
Crastnopol, Philip, *B.S., University of Maryland, 1938*..................New Jersey
Cusani, Benedict Albert, *B.S., Villanova College, 1939*....................Florida
Dalmáu, Miguel Sebastian, *B.S., University of Puerto Rico, 1939*.........Puerto Rico
Davis, William Joseph Graham, *A.B., University of North Carolina, 1939* District of Columbia
Diorio, John Daniel, *B.S., Mount St. Mary's College, 1939*...............Connecticut
Dunne, Thomas Benjamin, *A.B., The Johns Hopkins University, 1939*.......New York
Eaton, William Robert, *A.B., St. John's College, 1936*....................Maryland
Epperson, John Wallace Walker, *B.S., University of Maryland, 1941*......Maryland
Fowler, Richard Lowman, *B.S., The Citadel, 1938*...................North Carolina
French, Samuel Lawson, *A.B., American University, 1935*..................Maryland
Friedman, Paul Norman, *A.B., The Johns Hopkins University, 1939*.........Maryland
Garrison, Alfred Selman, *A.B., Western Maryland College, 1940*...........Maryland
Giglia, Tony Robert, Jr., *A.B., West Virginia University, 1939*.........West Virginia
Grave de Peralta, José Ignacio, *B.S., University of Maryland, 1939*............Cuba
Gray, David Benoni, *B.S., West Virginia Wesleyan College, 1938*........West Virginia
Hagan, William Baker, *B.S., University of Maryland, 1941*................Maryland
Hassler, Frank Stanley, III, *A.B., University of Pennsylvania, 1939*....Pennsylvania
Haydu, Joseph Frank, *Virginia Polytechnic Institute*..................West Virginia
Honigman, Alvin Herbert, *B.S., University of Maryland, 1939*.............Maryland
Hutson, Paul Gilliss, *B.S., University of Maryland, 1941*................Maryland
Koleshko, William Nickifor, *B.S., Washington College, 1939*.............New Jersey
La Mar, Robert Charles, Jr., *Goshen College*..............................Maryland
Lewis, Richard Quarles, *A.B., Duke University, 1939*......................Maryland
Livingstone, Robert Charles, *A.B., West Virginia University, 1939*..West Virginia
Lukats, Paul George, *University of Akron*....................................Ohio

MacDonald, Charles Renwick, *B.S., University of Maryland, 1941*.........Maryland
Matchar, Joseph Charles, *A.B., The Johns Hopkins University, 1939*.......Maryland
McMillan, Marcy Emory, Jr., *West Virginia University*...............West Virginia
Mele, Vincent James, Jr., *B.S., Seton Hall College, 1937*.................New Jersey
Méndez, Néstor Hernan, *B.S., University of Puerto Rico, 1939*...........Puerto Rico
Miller, James Delmar, *A.B., Ohio State University, 1939*......................Ohio
Minervini, Robert Virginius, *A.B., Columbia University, 1938*............New York
Musnick, Henry, *A.B., The Johns Hopkins University, 1939*................Maryland
Myers, Joseph Carl, A. B., *Western Maryland College, 1939*.................Maryland
Nash, Kenneth Powell, *University of California*........................California
Neff, Charles Amos, *A.B., Goshen College, 1938*............................Illinois
Parés, Maria Amalia, *B.S., University of Puerto Rico, 1939*..............Puerto Rico
Perez, Enrique, *A.B., New York University, 1939*.......................Puerto Rico
Peterson, Preston Horsley, *University of Utah*.............................Utah
Queen, Joseph Emmett, *A.B., Loyola College, 1939*........................Maryland
Rangle, Raymond Veto, *B.S., University of Maryland, 1941*................Maryland
Renshaw, Josephine Elizabeth, *B.S., University of Wisconsin, 1932, M.S., Wellesley College, 1933*...Maryland
Richards, Granville Hampton, *A.B., Franklin and Marshall College, 1939*..Maryland
Robbins, Martin Albert, *A.B., The Johns Hopkins University, 1939*........Maryland
Rosenstein, Louis Nathan, *B.S., University of Maryland, 1939*.............Maryland
Royer, Earl Linwood, *The Johns Hopkins University*......................Maryland
Rude, Richard Sprogoe, *Washington and Lee University*..................New Jersey
Sacks, Seymour, *A.B., The Johns Hopkins University, 1939*................Maryland
Samuels, Irving Leonard, *A.B., Duke University, 1939*....................Maryland
Sharp, Nathaniel, *University of Maryland, Loyola College*.................Maryland
Sigler, John Wiltshire, *A.B., Ohio State University, 1939*.....................Ohio
Soler-Favale, Marta Emilia, *B.S., University of Puerto Rico, 1939*.......Puerto Rico
Spier, Andrew Allan, *A.B., The Johns Hopkins University, 1939*............Maryland
Stafford, Harold Rellinger, *Juniata College*...........................Michigan
Stewart, Edwin Harvey, Jr., *The Johns Hopkins University*...............Maryland
Stier, Howard William, *A.B., The Johns Hopkins University, 1939*........New Jersey
Stoner, James Ernest, Jr., *A.B., Western Maryland College, 1939*...........Maryland
Taylor, Irving Julian, *A.B., The Johns Hopkins University, 1939*...........Maryland
Torres, José Manuel, *B.S., Manhattan College, 1939*....................Puerto Rico
Trader, Charles Weldon, *A.B., Western Maryland College, 1939*...........Maryland
Tunney, Robert Boone, *B.S., Loyola College, 1939*........................Maryland
Van Lill, Stephen Joseph, III, *A.B., Duke University, 1938*................Maryland
Varhol, Joseph Gregory, Jr., *B.S., Rutgers University, 1939*..............New Jersey
Wachsman, Irvin Louis, *University of Maryland, Johns Hopkins University*...Maryland
Walker, Samuel Haywood, *B.S., Davidson College, 1939*.............North Carolina
Warren, Frank Orville, Jr., *University of New Hampshire*...........New Hampshire
Webster, Thomas Clyde, *University of Maryland, Loyola College*............Maryland
Weiss, Maurice Richard, *University of Pittsburgh*.....................Pennsylvania
Wich, Joseph Carlton, *University of Maryland, Loyola College*.............Maryland
Wilson, Thomas Leslie, *University of Maryland*..........................Maryland
Wise, Robert Edward, *University of Pittsburgh*........................Pennsylvania
Woody, Arthur Overton, *Duke University*...............................Maryland
Worgan, David Kuykendall, *B.S., University of Maryland, 1941*...........Maryland
Yurko, Leonard Emory, *A.B., West Virginia University, 1939*..........West Virginia

FIRST YEAR CLASS 1940–1941

Acton, Elizabeth, *A.B., American University, 1940*......................New Jersey
Baker, Maurice Emmett, *A.B., Loyola College, 1940*.......................Maryland
Baldwin, Ruth Workman, *University of Maryland*...........................Illinois
Ballard, William Riley, Jr., *A.B., West Virginia University, 1940*......West Virginia
Bennett, Lillian Feykert, *A.B., M.A., University of California, 1938, 1939*..California
Berry, Herbert Lee, *University of Southern California*......................Arizona
Bitsack, Joseph William, *A.B., The Johns Hopkins University, 1940*.......New Jersey
Brandt, Frederick Bertram, *University of Maryland*...........District of Columbia
Brobst, Henry Thomas, *A.B., Oberlin College, 1940*....................Pennsylvania
Brown, Charles William, *A.B., West Virginia University, 1940*.........Pennsylvania

Brown, James Mack, *A.B., University of Alabama, 1940*	Alabama
Cappeto, Americo Peter, *B.S., St. Peter's College, 1940*	New Jersey
Colon-Yordan, Ernesto, *A.B., University of Puerto Rico, 1940*	Puerto Rico
Cook, Elmer Ellsworth, Jr., *University of Maryland*	Maryland
Corpening, William, *A.B., University of North Carolina, 1940*	North Carolina
Crosby, Robert MacGonigle Nelson, *The Johns Hopkins University*	Maryland
Curtiss, Robert Kimber, *A.B., Colgate University, 1940*	New York
Dann, Alfred Henry, *A.B., The Johns Hopkins University, 1940*	Maryland
Day, Edward Colson, *A.B., College of Wooster, 1939*	New York
Devers, William Joseph, *B.S., Villanova College, 1940*	Pennsylvania
Dillon, Harold, *B.S., University of Maryland, 1940*	Maryland
Dorman, Hamilton Peacock, *B.S., Bates College, 1940*	District of Columbia
Doyle, John Justin, *B.S., Boston College, 1940*	Massachusetts
Ehrlich, Daniel, *A.B., The Johns Hopkins University, 1940*	Maryland
Feldman, Lester, *A.B., Bucknell University, 1939*	Connecticut
Ferri, Henry Guy, *B.S.,University of Pittsburgh, 1940*	Pennsylvania
Finegold, Aaron Nathan, *B.S., University of Pittsburgh, 1940*	Pennsylvania
Frye, Augustus Homer, Jr., *B.S., University of Georgia, 1939*	Georgia
Galitz, Eli, *A.B., New York University, 1939*	New York
Garrett, Richard Mitchell, *Emory University*	Alabama
Gubnitsky, Albert, *B.S., University of Maryland, 1940*	Maryland
Guyther, Joseph Roy, *University of Maryland*	Maryland
Harris, William Myrick, *West Virginia University*	Maryland
Haught, John Stevenson, *West Virginia University*	West Virginia
Iguina-Jimenez, Manuel Antonio, *University of Puerto Rico*	Puerto Rico
Ingenito, Gabriel Andrew, *A.B., University of Connecticut, 1940*	Connecticut
Ingram, Charles Hal, *A.B., Duke University, 1940*	North Carolina
Isales, Luis Manuel, *B.S., University of Puerto Rico, 1939*	Puerto Rico
Jaworski, Melvin Joseph, *B.S., University of Maryland, 1940*	Maryland
Johnson, Wallace Riddick, *B.S., Wake Forest College, 1940*	North Carolina
Keeney, Dan Franklin, *University of Maryland*	Maryland
Kemper, Charles Alexander, *A.B., Duke University, 1940*	Maryland
Kinney, Earl Ray, *A.B., University of Kansas, 1940*	West Virginia
Kleiman, Allen, *A.B., The Johns Hopkins University, 1940*	Maryland
Latimer, Clarence Vinette, Jr., *A.B., Duke University, 1940*	New York
Livingstone, Stanley Hamilton, *A.B., West Virginia University, 1940*	West Virginia
Lurting, Frederick Wilbur, *B.S., University of Pittsburgh, 1940*	Pennsylvania
Mamula, Peter, *A.B., West Virginia University, 1940*	West Virginia
Markowitz, Arnold Robert, *A.B., West Virginia University, 1940*	West Virginia
McCormack, Lloyd Leo, *University of Virginia*	New Jersey
McFadden, Robert Burns, *B.S., Loyola College, 1940*	Maryland
McGrath, William Edward, Jr., *A.B., Loyola College, 1939*	Maryland
McMullin, Joseph Frederick, *A.B., Ohio University, 1938*	Pennsylvania
Meade, DeVoe Kepler, *B.S., University of Maryland, 1940*	Maryland
Miranda, Angel Neftali, *University of Puerto Rico*	Puerto Rico
Morgan, Jack Calvin, *A.B., West Virginia University, 1940*	West Virginia
Muth, Helen Tatum, *A.B., Connecticut College, 1927*	Pennsylvania
Myers, Myron Joseph, *A.B., The Johns Hopkins University, 1940*	Maryland
Nelson, Alfred Turner, *University of Michigan*	Maryland
Noll, Gilbert Edward, *B.S., Villanova College, 1940*	Pennsylvania
Ozazewski, John Casimir, *B.S., Loyola College, 1940*	Maryland
Palese, John Michael, *University of Maryland*	Maryland
Peters, Phillip John, *A.B., Marietta College, 1940*	West Virginia
Peters Robert Joseph, *West Virginia University*	Pennsylvania
Pfeil, Edgar Thornton, *University of Maryland*	Maryland
Pinas, Samuel Ronald, *B.S., University of Maryland, 1940*	Maryland
Pomeroy, William Henry, II, *B.S., Trinity College, 1938*	Connecticut
Raffucci-Arce, Francisco Luis, *University of Puerto Rico*	Puerto Rico
Range, James Jacob, *A.B., Duke University, 1940*	Tennessee
Ratliff, Cliff, Jr., *A.B., Duke University, 1940*	North Carolina
Ream, Norman, B., *B.S., Franklin and Marshall College, 1940*	Pennsylvania
Recht, John Munn, *A.B., Gettysburg College, 1940*	New Jersey
Rinehart, Arthur Middleton, *B.S., Trinity College, 1940*	Maryland

Robertson, Merritt Ezekiel, *A.B., Tusculum College, 1940*	North Carolina
Rogers, George Carraway, *A.B., University of North Carolina, 1939*	North Carolina
Rogers, William Brannon, Jr., *B.S., University of Akron, 1940*	Ohio
Santiago, Stevenson Parker, *A.B., Maryville College, 1940*	Puerto Rico
Sapareto, Rocco Louis, *B.S., Tufts College, 1939*	Massachusetts
Sauerman, Edward Edgar Karl, Jr., *University of Maryland*	Maryland
Scherlis, Irving, *A.B., The Johns Hopkins University, 1940*	Maryland
Scott, Wilbur Moate, *University of Georgia*	Georgia
Secskas, Violet Gertrude, *A.B., Flora Stone Mather College of Western Reserve University, 1940*	Connecticut
Shipley, Frank Mollman, *A.B., Western Maryland College, 1940*	Maryland
Shortle, James Samuel, *B.S., University of New Mexico, 1939*	New Mexico
Sones, Frank Mason, Jr., *A.B., Western Maryland College, 1940*	Maryland
Sterling, Harold, *B.S., University of Maryland, 1940*	District of Columbia
Strobel, Martin Edward, *A.B., Gettysburg College, 1940*	Maryland
Summerlin, Glenn Olson, *Emory University*	Florida
Thompson, Talmadge Stanley, *University of Maryland*	Maryland
Trent, William Franklin, *University of Tennessee*	Tennessee
Ubides, Pedro Federico, *B.S., University of Maryland, 1940*	Puerto Rico
Van Hook, Claude Ashton, Jr., *B.S., Villanova College, 1940*	New Jersey
Vargas, Dharma Luz, *B.S., University of Puerto Rico, 1940*	Puerto Rico
Waldron, Grayson Spencer, *A.B., University of North Carolina, 1940*	North Carolina
Walkup, Harry Ernest, *West Virginia University*	West Virginia
West, George Brooks, Jr., *B.S., College of William and Mary, 1940*	Virginia
Wheeler, John Edward, *B.S., Platteville State Teachers College, 1939*	Wisconsin
Will, David Reid, *Ohio State University*	Ohio
Williams, Thomas Richard, Jr., *A.B., Duke University, 1940*	North Carolina
Ziegler, Paul Randall, *University of Maryland*	Maryland

STUDENTS IN ART AS APPLIED TO MEDICINE

John T. Stringer, Jr.	Maryland
Elizabeth S. Cone	Maryland
Caroline H. Towson	Maryland
Virginia Woodland	Maryland
Ruth Cordish	Maryland

SUMMARY OF STUDENTS, 1940–1941

Medical Students	*Men*	*Women*	*Total*
Senior	83	9	92
Junior	84*	4	88*
Sophomore	85	3	88
Freshmen	94	6	100
	346	22	368
Withdrawal *1	1	0	1
	347	22	369
Students in Art as Applied to Medicine	1	4	5
	348	26	374

GEOGRAPHICAL DISTRIBUTION OF STUDENTS, 1940–1941

Alabama	2
Arizona	1
California	4
Connecticut	12
Delaware	1
District of Columbia	5
Florida	4
Georgia	5
Illinois	3
Indiana	1
Maryland (Baltimore 88)	158
Massachusetts	4
Minnesota	1
Mississippi	1
New Hampshire	1
New Jersey	22
New Mexico	1
New York	19
North Carolina	20
Ohio	6
Oregon	1
Pennsylvania	28
Rhode Island	1
South Carolina	1
Tennessee	3
Utah	2
Virginia	1
Washington	1
West Virginia	33
Wisconsin	1
Foreign	
Cuba	1
Puerto Rico	25

LIST OF COLLEGE SOURCES OF STUDENTS ENROLLED DURING 1940–1941*

Akron University	2
Alabama Polytechnic Institute	2
Alabama, University of	1
Alliance Junior College	1
American University	2
Antioch College	2
Bates College	1
Beckley Junior College	1
Bethany College	1
Blue Ridge College	3
Bridgewater College	2
Boston College	1
Boston University	1
Brigham Young University	1
Brooklyn College	2
Bucknell University	2
California, University of	4
Campbell College	1
Centenary Junior College	1
Central Y.M.C.A. College, Chicago	1
Chattanooga, University of	1
Chicago, University of	1
Citadel, The	3
Colgate University	1
Columbia Junior College	2
Columbia University	4
Connecticut, University of	2
Cornell University	3
Davidson College	2
Dickinson College	2
Duke University	15
Duquesne University	1
East Tennessee State Teachers Coll.	1
Elon College	1
Emory University	3
Erskine College	1
Flora Stone Mather College	1
Florida Southern College	1
Florida State College	1
Fordham University	1
Franklin and Marshall College	2
Georgetown University	1
George Washington University	2
Georgia, University of	4
Gettysburg College	3
Goshen College	2
Goucher College	1
Harvard College	6
Haverford College	1
Holy Cross College	1
Hunter College	1
Illinois, University of	1
Indiana University	1
Johns Hopkins University	54
Juniata College	2
Kansas, University of	1
Kent State College	1
La Salle University	1
Lenoir Rhyne College	1
Lewis Institute	1
Louisville, University of	1
Loyola College	20
Loyola University	1
Manhattan College	1
Mansfield State Teachers Coll.	1
Marietta College	1
Marshall College	1
Mars Hill College	2
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Maryland, University of	74

* Totals exceed student total because of multiple college attendance.

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McGill University	1
Meredith College	1
Miami University	1
Michigan, University of	1
Middlesex Junior College	1
Minnesota, University of	1
Mount St. Mary's College	1
Muhlenberg College	1
New Hampshire, University of	1
New Mexico, University of	1
New River State College	2
New York, College of the City of	1
New York University	10
Niagara University	1
North Carolina, University of	11
Northwestern University	1
Notre Dame, University of	2
Oak Ridge Military Institute	2
Oberlin College	1
Ohio State University	3
Ohio University	1
Oregon, University of	1
Pennsylvania, University of	4
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Portland, University of	1
Potomac State College	3
Puerto Rico, Polytechnic Inst. of	1
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Rhode Island State College	1
Rochester, University of	1
Rutgers University	1
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St. Peter's College	1
San Mateo Junior College	1
Santa Monica Junior College	1
Seton Hall College	1
Smith College	1
South Carolina, University of	1
Southern California, University of	3
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Stanford University	1
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Transylvania College	1
Trinity College	2
Tufts College	3
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Whitman College	1
William and Mary, College of	2
Wilson College	1
Wisconsin, University of	1
Yale University	1

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Although it was known at the time of Pauli (1) that thiocyanates produced a fall in blood pressure, it was not until a quarter of a century later that any extensive investigation was undertaken to determine the clinical value of this drug. During the period between 1925 and 1932 numerous reports appeared in the literature describing more or less variable results. Nichols (2), Smith and Rudolph (3), Palmer, Silver, and White (4), Fineberg (5), Saleeby (6), and Goldring and Chasis (7) reported a substantial reduction, more than 30 mm. of mercury, of the systolic pressure with relief of symptoms in 31 per cent to 90 per cent of a total of 349 cases. Toxic or untoward effects were noted in 17 per cent to 50 per cent of the cases. Because of the variable number of beneficial results and the large percentage of toxic reactions, interest waned and the number of cases reported diminished.

In 1936 Barker (8) renewed interest in the subject by reporting cases in which the dosage, blood level and toxic manifestations were correlated. He noted that the dose required to maintain a blood-thiocyanate level of 6 to 12 mg. per 100 cc. ranged from 0.15 gm. to 1 gm. per day. With such doses serious toxic effects were rare and beneficial results were evident in more than 50 per cent of the cases studied.

In 1939 Robinson and O'Hare (9) studied seventy-five hypertensive patients receiving the drug. The dosage was carefully controlled by frequent blood cyanate studies. In 63 per cent of the cases an average reduc-

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TABLE 1

CASE NO.	AGE	KNOWN DURATION	SYMPTOMS	HEART SIZE	ELECTROCARDIOGRAPHIC FINDINGS	RETINAL EXAMINATION	PHENOLSULPHONPHTHALEIN TEST, TWO HOUR EXCRETION	AVERAGE BLOOD PRESSURE BEFORE TREATMENT	AVERAGE BLOOD PRESSURE AFTER TREATMENT	BLOOD CYANATE IN MG. %	AVERAGE DAILY DOSE IN GRAINS	RELIEF OF SYMPTOMS	TOXIC EFFECTS
1	42	3 yr.	Dizziness, headache	Slight enlargement	Myocardial damage	Exudates	40	225/140	182/122	6.5	15	Yes	None
2	46	1 yr.	Dyspnea, headache	Moderate enlargement	Myocardial damage	Normal	50	220/112	180/104	7	12	Yes	None
3	54	1 yr.	Dyspnea, headache	Normal	Left axis deviation	Tortuous arteries	68	240/120	180/100	9.5	15	Yes	Slight dizziness
4	58	4 yr.	Dyspnea, substernal pain	Slight enlargement	Left axis deviation	Normal	36	210/130	150/100	12.5	9	Yes	None
5	49	3 yr.	Dyspnea, substernal pain	Slight enlargement	Left axis deviation	Narrow arteries	30	214/130	150/100	15	6	Yes	Dizziness, cheilosis
6	56	5 mo.	None	Normal	First degree block	Fundi normal	70	190/112	202/122	3.2	9	No	None
7	51	1 yr.	Dyspnea, substernal pain, headache	Moderate enlargement	Normal	Narrow arteries	50	230/120	180/80	12	9	Yes	None
8	51	2 yr.	Dyspnea	Normal	Myocardial damage	Narrow arteries	60	220/110	180/96	8.7	15	No	None
9	42	2 yr.	Dyspnea, headache	Moderate enlargement	Left axis deviation	Arteriovenous compression, exudates	90	252/130	212/114	7	12	Yes	None
10	50	13 yr.	Dyspnea	Moderate enlargement	Left axis deviation, myocardial damage	Narrow arteries	40	220/110	180/100	7	9	No	None. Blood pressure the same after discontinuance of cyanate

11	58	3 yr.	Dyspnea, headache, precordial pain	Marked enlargement	Old anterior infarct	Narrow arteries	60	220/120	180/110	5	12	Yes	None
12	37	4 mo.	Headache, dyspnea	Moderate enlargement, dilated aorta	Left axis deviation	Exudates	48	220/130	208/120	9	9	No	None
13	39	1 yr.	Headache	Moderate enlargement	U-waves	Normal	68	240/140	190/100	7	9	Yes	None
14	51	2 yr.	Headache, dyspnea	Slight enlargement	Left axis deviation, myocardial damage	Normal	60	220/120	160/100	15	9	Yes	Abdominal pain
15	45	2 yr.	Headache, dyspnea	Slight enlargement	Left axis deviation, myocardial damage	Normal	40	180/110	160/96	8	9	Yes	Gastro-intestinal upset
16	46	3 yr.	Headache, dyspnea	Marked enlargement	Left axis deviation, myocardial damage	Normal	18	240/130	190/110	14	3	Yes	Painful subcutaneous nodules
17	59	4 yr.	Precordial pain	Moderate enlargement	Auricular extrasystoles	Normal	75	182/110	132/90	6	6	Yes	Gastro-intestinal upset
18	39	2 yr.	Headache, dyspnea	Normal	Left axis deviation	Normal	50	214/132	168/118	7.6	24	Yes	None
19	45	2 yr.	Headache, dyspnea	Normal	Normal	Normal	50	220/130	180/110	12	4.5	Yes	Cheilosis, loss of hair pigment
20	41	3 mo.	Headache, dyspnea	Moderate enlargement	Left axis deviation, myocardial damage	Narrow arteries, arteriovenous compression	30	280/170	210/120	13	3	Yes	None

tion in blood pressure of 40 mm. of mercury systolic and 20 mm. of mercury diastolic was observed; drops of over 100 mm. systolic and 35 mm. diastolic were observed in three cases.

In the light of these favorable reports it was decided to study a number of hypertensive patients in the cardiac clinic at the University Hospital. The plan of study was somewhat as follows: The age of the patient, the known duration of symptoms and of the hypertension were established insofar as was possible. Retinal examinations were made to determine the state of the arteries and the presence of exudates or hemorrhages. Teleoroentgenograms were taken to determine the presence and degree of cardiac enlargement. Electrocardiographic tracings were made to detect evidence of myocardial damage. Phenolsulphonphthalein tests were carried out to estimate the presence and extent of impairment of renal function.

Several blood pressure readings were made on each patient under similar circumstances at intervals of one week. Phenobarbital was administered in doses of $\frac{3}{4}$ to $1\frac{1}{2}$ gr. a day. Physical activities were restricted to a minimum. Treatment for cardiac failure was instituted when indicated. Blood pressure determinations were then made at intervals of one to two weeks for a period of observation lasting six to eight weeks.

Potassium thiocyanate was then administered in doses of 9 gr. per day for three days, followed by 6 gr. daily for four days, at the end of which time an estimation of thiocyanate in the blood was made. The dosage was then altered so that a sufficiently high blood level, 7 to 15 mg. %, could be maintained to achieve a maximum beneficial effect. At intervals of two to three weeks blood levels were determined, blood pressure readings and variations in symptoms were recorded, and dosages of thiocyanate were adjusted. During the first three months of the study the thiocyanate used was dissolved in syrup of wild cherry. For the remainder of the time enteric coated tablets were used. The latter were found to give fewer and less intense gastro-intestinal disturbances.

In general it was found that where the blood pressure was excessively high and where symptoms were maximal there was no significant change under a regime of barbiturate medication and restriction of physical activities. Table 1 summarizes the effects of thiocyanate medication.

In fifteen of the twenty cases studied there was a significant drop, 30 mm. or more systolic, in blood pressure and an amelioration of distressing symptoms. In seven cases toxic symptoms were noted. These consisted of dizziness, abdominal pain, nausea, painful subcutaneous nodules, fissures at the angles of the mouth, loss of hair pigment, and failure of superficial wounds to heal as rapidly as previously.

Several of the more interesting cases may be discussed at this point in some detail.

CASE REPORTS

Case No. 9: The patient, a colored female, age 42, was admitted on the medical service of the University Hospital because of progressive dyspnea and headache. During a period of observation the blood pressure averaged 250 mm. of mercury systolic and 130 mm. of mercury diastolic. A bilateral splanchnic resection was performed by Dr. T. B. Aycock. The greater, lesser, and least splanchnic nerves and the first lumbar ganglion were removed. During the postoperative period the blood pressure averaged about 150 mm. of mercury systolic and 90 mm. of mercury diastolic. After discharge from the hospital the patient was not heard from for a period of six months. She then appeared at the cardiac clinic with the complaint of a recurrence of dyspnea and headache. Her blood pressure at this time was 220 mm. of mercury systolic and 114 mm. of mercury diastolic. With enforced partial bed rest and barbiturates there was no significant drop in pressure or amelioration of symptoms. Upon maintaining a blood thiocyanate level of 7 to 12 mg. % on a daily dose of 12 to 15 gr. of potassium thiocyanate a sustained fall in blood pressure to 150 mm. of mercury systolic and 90 mm. of mercury diastolic was effected for the ensuing ten months. The patient was relieved of her dyspnea and experienced no toxic effects.

Case No. 16: A 46 year old colored female was seen in the cardiac clinic after suffering from headache and exertional dyspnea for three months. She had been aware of her hypertension for four years. A urologic examination revealed a bifid renal pelvis on the right and a bilateral hydronephrosis. There was marked cardiac enlargement and an electrocardiographic tracing showed evidence of myocardial damage. The phenolsulphonphthalein excretion was 18% in two hours. Her blood pressure while under observation was 240 mm. of mercury systolic and 130 mm. of mercury diastolic. A blood level of 14 mg. per 100 cc. was obtained with as little as 4 gr. of potassium thiocyanate per day. The blood pressure dropped to 190 mm. of mercury systolic and 110 mm. of mercury diastolic. The headache and dyspnea diminished somewhat, but the patient developed painful subcutaneous nodules which only disappeared several days after discontinuing thiocyanate medication. Resumption of thiocyanate therapy apparently caused a recurrence of the painful nodules so that the drug was withdrawn at the patient's request.

Case No. 19: A white male, age 45, first applied for treatment in November, 1939. He had been suffering from repeated attacks of violent frontal headaches, accompanied by vomiting, which lasted seven to eight hours and occurred three to four times a month since May, 1937. During most of this time he had been taking $1\frac{1}{2}$ gr. of phenobarbital and 10 to 15 gr. of aspirin a day, apparently without relief.

In 1920 a bilateral apical tuberculous infection was discovered. This was arrested during the ensuing six to seven years.

The patient's father and two sisters died of complications of hypertensive cardiovascular disease. A brother, age 37, was affected with the same ailment.

When first seen the blood pressure was 220 mm. of mercury systolic and 130 mm. of mercury diastolic; the heart was of normal size and there was no evidence of myocardial or renal damage; there were no gross retinal abnormalities.

From November, 1939 to May, 1940 the physical activities were restricted and several hours of enforced bed rest daily were obtained. Phenobarbital was continued. There was only a slight decrease in the frequency of the headaches. After three weeks' administration of $4\frac{1}{2}$ gr. of potassium thiocyanate daily the blood pressure dropped to 180 mm. of mercury systolic and 110 mm. of mercury diastolic. The blood level was 15.6 mg. per

100 cc. During the following three weeks as the blood level rose to 20 mg. per 100 cc. it was observed that the patient's beard, eyelashes and roots of the hair over the entire body became blond. Fissures developed at the angles of the mouth and the patient noted that superficial wounds healed much more slowly than heretofore. At this time the blood pressure was 158 mm. of mercury systolic and 104 mm. of mercury diastolic. The dosage was then adjusted so that a blood level between 10 and 15 mg. per 100 cc. was maintained. The newly grown hair then contained the normal amount of pigment, the fissures at the angles of the mouth disappeared, and the healing time of superficial wounds apparently returned to normal. At infrequent intervals the patient felt somewhat dizzy and was aware of a burning sensation in the epigastrium. From May, 1940 to April, 1941 he had no headache. The blood pressure averaged 178 mm. of mercury systolic and 106 mm. of mercury diastolic. He then abandoned the cyanate and was asymptomatic until four weeks later, when he developed a severe headache, nausea and vomiting which lasted eighteen hours. He then asked for a resumption of cyanate therapy.

Case No. 20: A white male, age 40, complained of exertional dyspnea and violent frontal headaches of three months' duration.

His personal and family history were noncontributory.

When first seen his blood pressure was 280 mm. of mercury systolic and 170 mm. of mercury diastolic. The heart was moderately enlarged; there was electrocardiographic evidence of myocardial damage; fundal examination revealed a hypertensive retinitis. There was rather marked impairment of renal function as demonstrated by the diminished excretion of phenolsulphonphthalein. Slight pretibial edema was evident.

The patient was put to bed, digitalized, and placed on a diet restricting fluids and salt. Phenobarbital was administered in daily doses of 1½ gr. After a month the dyspnea and edema disappeared and the blood pressure dropped to 266 mm. of mercury systolic and 158 mm. of mercury diastolic; however, the violent frontal headaches occurring three to four times a week persisted. He was then given 6 gr. of potassium thiocyanate daily. Within three weeks a blood level of 18.7 mg. per 100 cc. was obtained and the blood pressure was 218 mm. of mercury systolic and 128 mm. of mercury diastolic. He had only a few mild headaches. As the blood level rose to 28 mg. per 100 cc. he developed slight nausea and generalized weakness. The lowest blood pressure recorded at this level was 212 mm. of mercury systolic and 112 mm. diastolic. As the dosage was reduced to 3 gr. per day the nausea and weakness subsided and the blood pressure ranged from 220 to 230 mm. of mercury systolic and 120 to 130 mm. of mercury diastolic.

During the four months under cyanate therapy the patient suffered only one severe headache. His dyspnea was less noticeable. He complained occasionally of slight nausea.

Recently, additional reports seem to coincide in general with results obtained in this clinic. Griffith, et al. (10) reported successful treatment of fifty-nine out of 101 cases so observed. They noted that a successful result was obtained more often when the minute vessel pressure and cutaneous lymphatic flow were normal.

Blaney, et al. (11) observed twenty patients in which there was a satisfactory lowering of blood pressure in ten, with a complete remission in four of the cases. They recorded an optimum beneficial effect when the blood cyanate level was 8 to 15 mg. per 100 cc. Toxic effects were more common when blood levels higher than this were maintained but were also seen with levels as low as 6 mg. per 100 cc. It was their conclusion that the duration of hypertension, degree of cardiac and renal failure, presence of retinitis, and severity of symptoms gave no clue as to whether or not a beneficial response would be obtained.

Still more recently Lindberg, Wald, and Barker (12) reported on the pathologic effects of thiocyanate when the drug was given to dogs in toxic doses over a long period of time. They noted that there was a reduction in the erythrocyte count, hematocrit value, total serum proteins and plasma cholesterol, that the erythrocyte sedimentation rate was increased, and that there were significant liver and bone marrow changes. They emphasized the fact that there were no comparable changes reported in man when the drug was used in therapeutic doses.

COMMENT

From recent reports in the literature and from the experiences reported in table 1 certain features were apparent. Potassium thiocyanate when administered in therapeutic doses to hypertensive individuals appreciably reduced the blood pressure and diminished the severity of symptoms in a majority of patients so treated. Severe toxic manifestations appeared in only a small percentage of cases. The drug was effectual when usual measures were of no avail.

Beneficial effects and toxic manifestations could not be predicted with any regularity from an analysis of the clinical data prior to the onset of therapy. In general, however, it appeared that a maximum depressor effect was obtained with higher blood levels of thiocyanate, that amelioration of symptoms usually paralleled the degree of fall in blood pressure, that more toxic effects were evident when the blood cyanate was at a high level, and that high blood levels were reached more quickly and maintained when there was evidence of significant renal damage.

In the cases studied in the cardiac clinic there were significant variations in blood pressure, blood levels and in the degree of amelioration of symptoms with a constant daily intake of thiocyanate. The variations could be accounted for by variations in food and fluid intake, the degree of renal and cardiac decompensation, and by failure of the patient to fully cooperate.

It would seem that in potassium thiocyanate one has a potent therapeutic agent which might be used to relieve the distressing symptoms of hypertension when all other measures have failed. It is essential that the therapist have the fullest cooperation of the patient so treated and that almost constant observation be maintained. The therapeutic efficacy could then be ascertained for each case and serious toxic reactions could be avoided.

An argument for treating young hypertensive individuals who are asymptomatic but in whom symptoms are apparently imminent might be advanced on the grounds that postponement of the symptoms might be effected. This, of course, would be feasible only if there were no significant toxic effects. Since such effects cannot be predicted it would seem worth while in certain selected cases to consider a trial of the drug.

SUMMARY AND CONCLUSIONS

1. A series of twenty cases of hypertension treated with potassium thiocyanate is reported.
2. The significant lowering of blood pressure with an amelioration of symptoms is noted in fifteen of the cases.
3. Toxic effects are noted in six cases.
4. Continued use of the drug would seem to be indicated when distressing symptoms are not alleviated by ordinary procedures.
5. Careful constant observation is essential to the obtaining of a maximal beneficial effect.
6. Further clinical investigation would seem to be warranted on the basis of recent reports in the literature and upon the experiences presented in this paper.

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CHRONIC CACHEXIA SIMULATING SIMMONDS' DISEASE*†

A Case Report with Autopsy

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In recent years much light has been thrown on the activities of the pituitary gland. This is the result of the brilliant investigative work that has been done in the clinic and laboratory. The relations of this gland of internal secretion to the control of functions, such as growth and differentiation, and the influence of its hormonal activity upon the other endocrine glands have been demonstrated. It has been called upon occasion the "master" gland and also "the leader of the endocrine orchestra." In view of its dominant position, pathologic changes in the hypophysis have been considered to produce many varieties of disease syndromes among which is the so-called pituitary cachexia or Simmonds' disease. The term *cachexia* (caco–bad, hexis–habit) is a clinical expression which has been used in a rather loose way to designate bodily deterioration accompanied by the subjective feeling of exhaustion met with in a number of diseases and intoxications.

The anterior pituitary gland has also been demonstrated to be indispensable for the complex phenomenon of growth, although the mechanism by which the anterior pituitary gland makes possible the growth process and the chemical nature of the secretion which accomplishes this effect are in neither case fully understood. Pituitary dwarfism, according to Shelton (13), "is a chronic physiologic disorder having its origin in aberrant function of the pituitary gland during the period of somatic development and characterized by curtailment of the growth processes, with resultant small or diminutive stature and retention of other infantile characteristics."

The following case presents the clinical picture of pituitary cachexia and dwarfism without any evidence in the postmortem examination of a destructive lesion of the anterior lobe of the pituitary.

CASE REPORT

The patient was a young man twenty-four and a half years of age, whose complaint was that of weakness, loss of weight, anorexia, diarrhea, and stunted growth. His mother died from pulmonary tuberculosis when he was four and a half years old. As a child he had chickenpox, measles, mumps, and whooping cough. He grew and developed normally until the onset of his illness at the age of thirteen and a half, when he measured

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fifty-nine inches in height and weighed eighty-six pounds, both being just a little below the optimal for his age and sex.

The present illness began in March, 1929 with acute abdominal pain associated with fever, vomiting, diarrhea, exhaustion, and progressive loss of weight. Three months later his weight was reduced to sixty-one pounds. At that time he showed no developmental defects. The systolic blood pressure was 70 mm. hg. and the diastolic 40 mm. hg. He had a pulse rate of 108 and his temperature was normal. The skin was sallow, and the superficial lymph glands in the neck were enlarged. A soft systolic murmur was

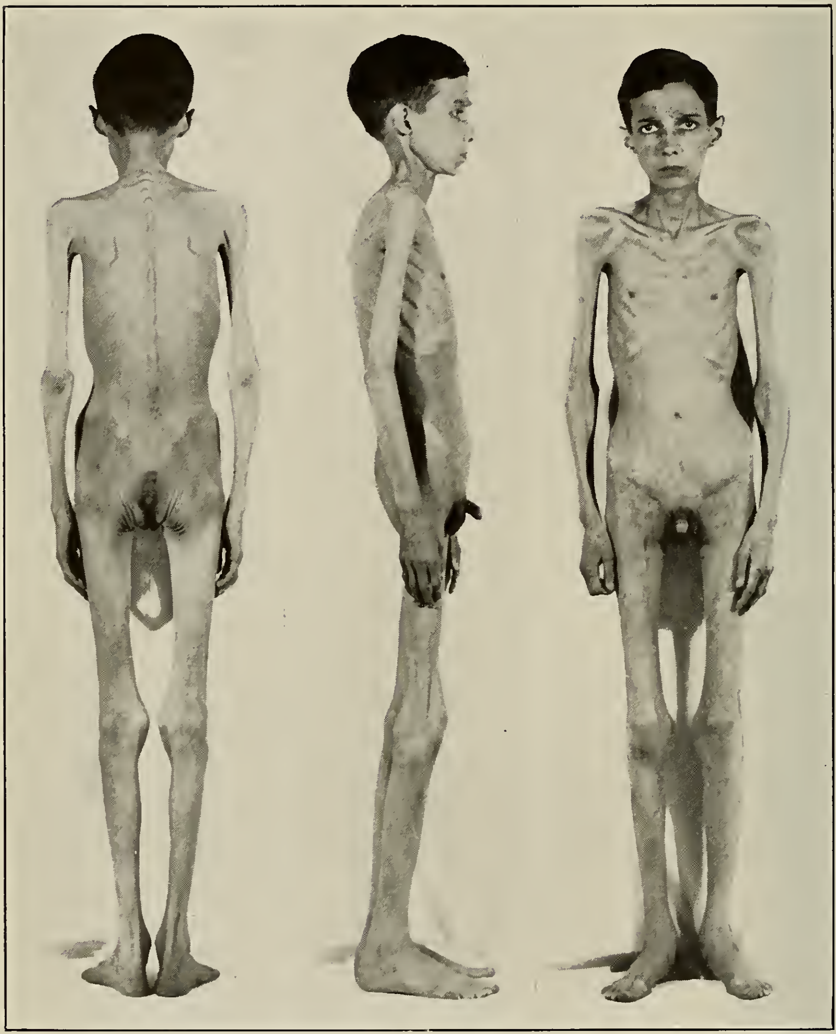


Fig. 1. View of the patient at twenty-three years of age. The extreme cachexia, infantilism, increased pigmentation, and genital hypoplasia may be noted.

heard at the base of the heart and there was some local tenderness in the right lower abdominal quadrant. A roentgenogram of the chest showed numerous healed miliary tubercles scattered throughout both lungs. In addition to his weakness and emaciation he continued to suffer periodically with attacks of diarrhea, vomiting, and abdominal pains for the next four years, when his health improved somewhat so that he was able to return to school for the ensuing three years, after which he had an exacerbation of his symptoms.

On April 21, 1938 he was admitted to the University Hospital. The records indicated that he was suffering from intractable diarrhea averaging from ten to twelve watery, blood-tinged movements daily, accompanied by occasional vomiting of blood. He was greatly emaciated and undersized and, although then over twenty-three years of age, he manifested none of the characteristic changes incident to puberty. The eyelids were edematous. There was a slight yellowish-brown pigmentation of the face and marked trophic skin changes in the extremities. The abdomen was distended and exhibited evidence of accumulated ascitic fluid; the superficial abdominal veins were prominent and the inguinal glands felt shotty.

Laboratory studies. The urinalysis and kidney function were normal. Examination of the blood showed hemoglobin 65%, red cells 3,864,000, white cells 5,980, and a normal differential count. The blood chemical examination was reported as follows: sugar 74 mg.%, chlorides 290 mg.%, phosphates 2.4 mg.%, calcium 9.8 mg.%, nonprotein nitrogen 28 mg.%. Total serum proteins were 3.52 grams per 100 cc., and the total 24 hour creatinine output was 0.150 grams. The serologic test for syphilis was negative. There was no reaction to .001 mg. old tuberculin. All agglutination tests were negative. No intestinal parasites were found and repeated stool examinations were negative for tubercle bacilli, as was also guinea pig inoculation. The stool contained 34.67% of total fat. The assay for female sex hormone by the castrated rat method (content of total specimen) showed less than three rat units. The estrin was not increased.

While in the hospital the patient was given three unit doses of insulin in the hope of stimulating his appetite. These caused insulin shocks and were discontinued. Ten per cent glucose was administered intravenously daily, and he had several blood transfusions to which he responded well. He was discharged from the hospital on May 30, 1938 as unimproved. A month later while in the clinic the following additional observations were made.

The patient presented the combined clinical features of dwarfism, infantilism, and severe cachexia. He was too weak to walk more than a few steps and had to be carried up and down the stairs.

He measured fifty-nine and three-fourths inches in height and weighed fifty-six pounds. This represented a growth of three-fourths of an inch and a weight loss of thirty-two pounds since the age of thirteen and a half years, or during a period of ten years. His height was the optimal for a boy of thirteen and a half years, and his weight the optimal for a boy eight and a half years of age. In relation to his height he was thirty-one pounds below optimal weight.

There was some mental retardation as well as physical underdevelopment. He had retained his juvenile voice and his interests were puerile. The skin was the color of *café au lait.* It was thin, dry and transparent so that the anatomic structures underneath, especially the veins, were distinctly visible. Below the knees the skin was rough, scaly and slightly fissured, and the color was purplish-red as a result of long-standing trophic edema. There were slight pigmentary changes which in color and size somewhat resembled freckles scattered over the entire body. A fairly good suit of fine hair on the scalp was uniformly distributed, except for an area of alopecia to the left of the vertex measuring 1½ cm. in width and 4 cm. in length. The eyebrows and eyelashes were normal.

There was no hair on the face, and hirci and crines pubis were absent. The nails were brittle and clubbed fingers were observed.

On account of the extreme wasting of the muscles and the absence of even a vestige of subcutaneous fat, the anatomic landmarks stood out prominently. There was marked genital hypoplasia. The penis was infantile; both testicles were the size of small olives and were descended. The scrotum was slightly swollen and edematous. There was a patch of vitiligo on the dorsum of the penis.

The face presented the appearance of precocious senescence. It was drawn, wrinkled and cachetic, and his expression suggested a depressed and melancholy mood. The teeth showed early decay, with infection and retained roots. There were no changes in the eye grounds; the pupils reacted sluggishly to light and accommodation. The tonsils were not infected. The thyroid was normal. There were a few small glands palpable in the neck. On account of the extreme emaciation, the ribs, shoulders, clavicles and scapulae disclosed well defined skeletal outlines. The lungs were clear on percussion and auscultation, and the heart was normal except for a faint, soft systolic murmur at the base.

The abdomen was somewhat distended and tympanitic; the abdominal wall was thin and the muscles rigid. There was marked splashing in the stomach. The liver did not appear to be enlarged; the spleen and other viscera were not palpable. There was trophic edema of both feet and ankles. All reflexes were hyperactive.

Additional laboratory studies showed normal urine. Fractional gastric analysis showed a maximum acidity of 70 degrees of hydrochloric acid and 77 degrees of total acidity at the end of ninety minutes. Fasting blood sugar was 106 mg.% and blood calcium 10.8 mg.%.

The basal metabolic rate was −28 per cent, but subsequently dropped to −32 per cent. An electrocardiogram showed sino-auricular tachycardia and low amplitude of the ventricular complexes.

Roentgenologic studies. The roentgenologic findings indicated the existence of a spastic shortened colon and healed miliary tuberculosis of the lungs. The pituitary fossa was quite small and flattened, and there was no erosion of the anterior or posterior clinoids or of the floor of the pituitary fossa. The roentgen examination of the bones showed a diminution in their size and a delay of fusion in many of the epiphyses.

Clinical course and treatment. From the history and physical findings it was felt that the patient had a severe endocrine dysfunction, probably a pituitary cachexia resulting from miliary tuberculosis. From June, 1938 to January, 1939, he was treated with whole anterior pituitary and APL extracts (antuitrin, antuitrin-S, antuitrin-G and anterior pituitary extracts), and preparation of the adrenal cortex (eschatin). This treatment was supplemented by large doses of vitamin concentrates to compensate for their loss through the severe diarrhea. He semed to respond to this therapy for a time with a gain of weight, an increase of strength, a lessening of diarrhea, and disappearance of the peripheral edema. There was a perceptible increase in his ability to engage in physical and mental activity. When admitted he was unable to walk without aid. In October, 1938, he felt better and was able to visit the clinic unaccompanied and walk a considerable distance. For three months during this period he was given $\frac{1}{2}$ gr. of thyroid every day, with a resultant elevation of the basal metabolic rate but without any other significant changes. In January, 1939 testosterone propionate (oretone) injections were begun and a remarkable change was noted. He became much stronger and cheerful, and opened a workshop at home where he engaged in manual activities. There was also an increase of muscle tone and an increase in penile size and erections. In August, 1939 the entire treatment was discontinued and within a short period there was a recurrence to his previous state. In November, 1939 treatment was resumed but his course from that time was slow and progressively downhill. On the day of his death he developed violent pains in the abdomen, with vomiting and diarrhea. He was given salt solution, glucose, and large doses of adrenal cortical hormone, but there was no improvement and he died on March 8, 1940.

Postmortem examination by Dr. D. J. Greiner: The body was that of an adult white male and measured 150 cm. in length. The entire body was markedly emaciated. Nothing of importance was noted in the scalp, eyes, ears, nose, and mouth. There were no masses in the neck. The ribs were prominent and the abdomen was flat. The external genitalia were of the infantile type.

The brain weighed 1400 grams and externally appeared normal. The pituitary gland was of normal size and somewhat firm in consistency; the sella showed no gross abnormality. The middle ears and nasal sinuses were normal. There was no subcutaneous fat over the chest and abdomen, and the musculature was thin. Both lungs were free and there was no pleural fluid. The pericardial sac contained approximately 4 cc. of straw-colored fluid. The heart was free. The abdominal organs were in normal position. The omentum contained very little fat and was bound to the colon near the ileocecal valve. In this region several loops of small bowel were adherent.

Lungs: The left lung weighed 200 grams. Its surface was pale gray and pink in color, with scattered dark gray shotty nodules varying from a pinhead to 3 mm. in diameter. On sectioning, the cut surface was pinkish-gray in color and the posterior portion of the lower lobe showed hypostatic congestion. There were a few scattered hard nodules in both lobes. The vessels and bronchi were normal. The right lung weighed 220 grams and its surface was the same as in the left. On sectioning, the cut surface was similar to the left and the lower lobe showed hypostatic congestion.

Heart: The heart weighed 150 grams. On sectioning, the muscle was of fair tone and pale brownish-red color. Its thickness measured .25 cm. for the right and .8 cm. for the left ventricle. The valve leaflets were negative and the circumference measured: tricuspid 9 cm., pulmonic 6.5 cm., mitral 8 cm., aortic 5.5 cm. The coronary ostia were patent and the vessel walls were normal on sectioning. The first portion of the aorta showed nothing of note.

Spleen: The spleen weighed 170 grams. The surface was roughened laterally by old fibrous adhesions.

Liver and gallbladder: The liver and gallbladder together weighed 1100 grams. The liver was somewhat soft but the cut edge did not evert.

Pancreas: The pancreas measured 17 cm. in length and showed nothing of importance.

Adrenals: Both adrenals were small and considerably flattened; on sectioning, they showed a very narrow pale cortical layer and a small amount of light gray medullary substance.

Kidneys: The kidneys together weighed 190 grams. The surface of the left kidney was smooth; on sectioning, the cortical tissue was not well demarcated. The vessels were congested; the capsule stripped with ease and left a smooth surface. The right kidney was similar to the left in every detail.

Urinary bladder: The urinary bladder was small, collapsed, and contained a scant amount of cloudy fluid.

Prostate: The prostate was infantile and grossly showed nothing of importance.

Testicles: The testicles were underdeveloped and soft in consistency; on sectioning, the tissue was wet, soft, and of a pale gray color.

Thyroid: The thyroid was small and showed a well developed isthmus. On sectioning, the glandular tissue had a pale brown glistening appearance.

Gastro-intestinal tract: The stomach and upper small bowel were filled and distended by semifluid material. At the junction of the middle and lower thirds of the ileum, the bowel wall was thick and discolored; the lumen was markedly narrowed and crossed by fibrous bands. There were two small openings, one into the terminal ileum and the other into the ascending colon. This portion of the bowel was surrounded by dense fibrous adhesions. The appendix showed nothing of note.

Anatomic diagnosis. Miliary tuberculosis, lungs (healed); chronic enteritis, ulcerative with ileocolonic fistulas (character undetermined); obstruction of terminal ileum; retarded development; emaciation; hemorrhage,—petechial, multiple, subependymal zone.

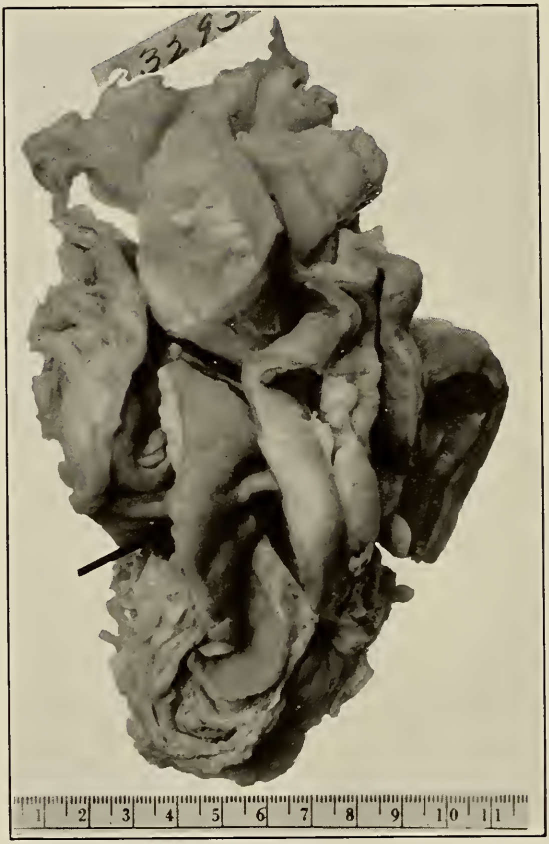


FIG. 2. Specimen showing the ileocolonic fistula as obtained by postmortem examination.

Microscopic notes. Lung: Several sections of lung demonstrated a congestion of the capillary bed which was considerable in some areas. There were also scattered areas of atelectasis in the lung. Calcified nodules and tubercles surrounded by dense fibrous tissue were seen in the section. The lumina of the bronchioles were empty but the interstitial tissue, particularly the peribronchial tissue, contained macrophages, anthracotic pigment and round cell infiltration.

Heart: The muscle fibers appeared small and an occasional individual fibre was hypertrophic. The small capillaries were filled with red blood cells. There was slight round cell infiltration in some areas.

Spleen: The splenic pulp was filled with red blood cells in most areas. Microscopically, the malpighian bodies were easily seen. The pulp in general appeared slightly increased in fibrous tissue.

Liver: The liver tissue showed considerable round cell infiltration most distinct about the portal triad areas. There was some slight fatty change in the tissue. The cellular reaction was of a nonspecific type.

Pancreas: The lobulated glandular tissue contained many prominent islets. There were two separate areas of degeneration which appeared fibrous in character and showed no inflammatory reaction or hemorrhage.

Kidney: The interstitial tissue and glomeruli appeared normal. Most of the convoluted tubules contained acellular granular debris. These were lined by intact epithelium.

Adrenal: The capsule was slightly thickened by fibrous tissue. The cortex was thin but aside from reduction in size the individual zones were easily seen. There was a benign hyperplasia of the medullary tissue which was somewhat vascular.

Intestine: The mucosa showed surface ulceration and sloughing. The wall in this area was fibrous and infiltrated by chronic inflammatory cells. This reaction extended to and involved the roughened peritoneal surface.

Thyroid: Most of the acini were of moderate size and contained colloid. There was no lymphoid tissue present and only a small amount of fibrous tissue. One group of immature acini was found on one margin.

Prostate: The stroma showed increased fibrous tissue and supported groups of acini, in some of which was an overgrowth of lining epithelium.

Testicle: The testicular tissue was infantile in type. The tubules showed no evidence of spermatogenesis and were supported by a loose fibrous stroma. The epididymis showed nothing of note.

Node: A section of node showed only benign inflammatory hyperplasia.

Gross description by Dr. W. W. McKinney: Brain: The hemispheres were symmetrical. There was slight tendency to microgyria in the left occipital lobe. No areas of softening or hemorrhage were seen. The structures at the base appeared normal and the vascular system was normal. There was a very slight pressure cone. Sections through the pons, cerebellum, and medulla were grossly normal. The hypophysis measured 6 by 15 by 5 mm. On sectioning, the gland had a uniform structure. There were no areas of softening, hemorrhage, or necrosis seen grossly. On its external surface the gland was smooth and grayish in color.

Microscopic notes by Dr. James G. Arnold, Jr.: Block 1—Hypophysis: This section was taken transversely through the hypophysis at the level of the stalk so that the section did not extend to the posterior lobe. A small portion of the stalk was present and normal. The structure of the anterior lobe was uniform. There seemed to be a normal distribution between chromophil and chromophobe cells. At the base of the stalk there were a dozen or more acini distended with colloid. The hypophysis both grossly and microscopically showed no deviation from normal.

Sections through the basal ganglia at the level of the left chiasm showed that the superior convolutions of the right frontal and pons were normal except for several petechial hemorrhages in the subependymal region.

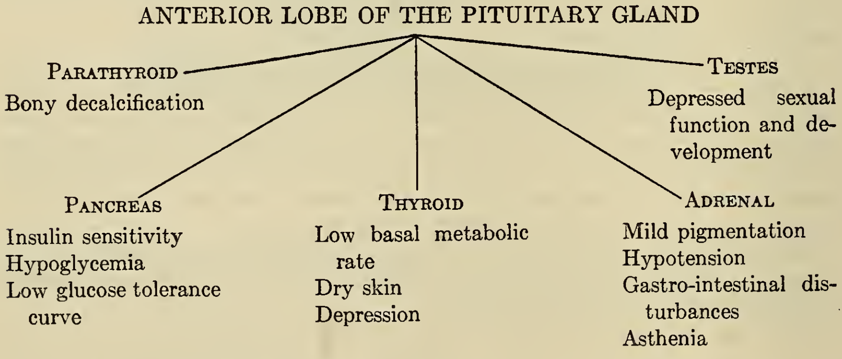
Diagnosis. Hemorrhage, petechial, multiple, subependymal zone.

DISCUSSION

The case presented herewith offers many interesting but puzzling problems to the investigator who wishes to interpret properly the pathologist's report and the relationship of the pathologico-anatomic findings to the clinical signs and symptoms. In reviewing the clinical records one is impressed by the close resemblance to Simmonds' syndrome. Without the postmortem examination, in which a small but histologically normal pituitary was found, this case could readily have been placed into the small group thus far reported as Simmonds' disease.

It is instructive before further discussion to review briefly the physiology of the anterior pituitary lobe. Extensive investigation has repeatedly revealed the profound effect of hypophysectomy on the other endocrine glands. In experimental animals following pituitary ablation dwarfism, atrophy of the thyroid, gonads and adrenals have been noted. In addition, a decrease of metabolism, hypoglycemia, increased insulin sensitivity, lowered resistance to cold, and decrease in weight of the body organs (liver, kidneys and spleen) follow removal of the anterior lobe of the pituitary. The tremendous influence of this gland would accordingly be expected to result in strikingly important clinical phenomena whenever its tissue is involved by pathologic processes. As a matter of fact, this does not always hold true since there are reported cases in which extensive destruction of the anterior pituitary has occurred without subsequent clinical changes. It has been demonstrated that certain variations in secretory function are associated with changes in function of the various cellular components (acidophils, basophils, and chromophobes) of the anterior lobe. Although great strides have been made the stage has not yet been reached where the actual production of a certain hormone can be assigned to a specific cell.

For simplification of the clinical aspects of the effects of the anterior pituitary on the other endocrine glands as observed in the case herewith reported, the following diagram is presented.



ANTERIOR LOBE OF THE PITUITARY GLAND

PARATHYROID
Bony decalcification

TESTES
Depressed sexual function and development

PANCREAS
Insulin sensitivity
Hypoglycemia
Low glucose tolerance curve

THYROID
Low basal metabolic rate
Dry skin
Depression

ADRENAL
Mild pigmentation
Hypotension
Gastro-intestinal disturbances
Asthenia

It is quite evident that organic destruction or toxic changes must result in absence or variation in the effect of the thyrotropic, gonadotropic, adrenotropic, parathyrotropic, diabetogenic, and growth hormones.

However, in the case report there is no evidence of pituitary change even though there is almost perfect correlation in symptomatology with Simmonds' syndrome, and therefore one is led into the realm of speculation as to the mechanism of production of the observed signs and symptoms of definite pituitary origin.

Briefly, the cardinal clinical features of Simmonds' disease are extreme cachexia, premature senility, loss of body hair, mental disturbances, low basal metabolic rate, hypotension, and microsplanchnia. In addition, changes in the integument occur, including falling of the teeth and hair, particularly in the axillary and pubic regions, trophic changes in the nails, and thickening and loss of lustre of the skin. There is a general muscular weakness and debility, and a corresponding atony of the gastro-intestinal tract, which lead to such symptoms as constipation, vomiting and achylia gastrica. There are virtually always changes in the sexual function amounting in the male to complete impotence and loss of libido.

Many of those who have contributed to the knowledge of Simmonds' disease have postulated that a proved case must show organic changes in the anterior pituitary lobe. The pituitary may be involved by new growths, tuberculosis, syphilis, cysts, fractures of the skull, postpartum embolism, and metastatic abscesses. Most cases have been reported in women, especially following repeated childbirths.

From time to time, however, there have appeared in the literature cases of undoubted pituitary cachexia with relatively normal findings on examination of the hypophysis. In a recent article Doane, Blumberg and Teplick (6) present evidence that Simmonds' syndrome may be of functional origin. They report that "The pituitary can be completely inactive functionally although normal in growth and histological appearance." They cite reports of cases by Plummer and Jeager, Samet-Mandels, and DeLaure and de Gennes, in which the hypophysis was found to be normal at autopsy although subject to external pressure in the first two instances.

It has been felt for some time that the eosinophilic cells of the pituitary have some relation to the Simmonds' syndrome and that, as in acromegaly, the eosinophilic tissue is known to be hyperactive. It is readily possible to postulate a deficiency in the secretion of these cells when a strikingly opposite pathologic picture is discovered. Brown cites a number of cases of Simmonds' syndrome in which the destruction or absence of the eosinophilic cells was noted. Other observers have also reported on this possibility.

Kunstadter (10) writes that "It is conceivable that a suppression of the anterior lobe may occur as a result of a congenital defect, a toxic influence, secondary to the infectious diseases of childhood, or it may be due to a functional exhaustion resulting from the added strain of puberty, or a combination of any of these factors." He gives impressive evidence to prove his statement. It is also believed to be a functional exhaustion of the pituitary gland which accounts for the hypophysial cachexia following repeated labors. A number of observers (Herrick, Riese, Maresch, May, and Robert) present evidence favoring this possibility. There exists, therefore, impressive combined clinical and research evidence that pituitary cachexia may result from functional as well as organic changes in the pituitary.

That this situation obtains with reference to other endocrine syndromes is well known and acceptance of this belief goes a long way to explain the clinical phenomena observed in the case report accompanying this article. It is of extreme importance to note that in this patient there was a combination of a chronic debilitating disease (miliary tuberculosis) with a severe nutritional deficiency resulting from chronic diarrhea, in turn caused by an ulcerative enteritis and an ileocolonic fistula. This leads to a discussion of (1) the rôle of miliary tuberculosis in the production of pituitary emaciation, and (2) infantilism resulting from chronic enteritis. It has been recognized for some time that there is a common association between tuberculosis of some degree and Simmonds' syndrome. Tuberculosis is not an uncommon disease of the pituitary. In 1914 Simmonds reported a whole series of cases of tuberculosis of the hypophysis. Altman, in 1928, stressed the relationship of Simmonds' disease to pulmonary tuberculosis. The symptoms of chronic tuberculosis in some instances may simulate pituitary cachexia. This is probably the result of a toxic condition that may affect the anterior pituitary, as suggested by Kunstadter (10). Osgood (11) reported a case presenting a complete clinical and laboratory picture of pituitary cachexia with a normal pituitary gland in which tuberculosis was discovered years after the original study. The studies of Plaut have revealed the interesting fact that in cachexia caused by tuberculosis the basal metabolic rate is characteristically elevated, whereas in pituitary cachexia it is markedly lowered. This observation is an important differential diagnostic point. The recession of secondary sex characteristics so marked in Simmonds' disease is usually not found in tuberculous cachexia. It is probable in the reported case that the miliary tuberculosis played some part in the induction of the chain of symptoms which followed, since this patient was known to have had his onset of illness in 1929 and grew progressively worse from that time. At autopsy, healed miliary tubercles were found in both lungs. This was proved microscopically.

The possibility of infantilism in the reported case, following a chronic debilitating disease and nutritional deficiency, raises once again a most interesting question, particularly since the rôle of lack of minerals and vitamins is now better realized. Infantilism has been reported as a sequel to a number of disorders. Davidson (5) reported infantilism in chronic ulcerative colitis and discussed the mechanism of its production. He indicated that secondary infantilism may occur in chronic diarrhea because the growing organism is deprived of substances needed for the normal maintenance, maturation and growth. Infantilism occurs most strikingly in patients who are ill during the period of physical and sexual maturation. There is a retardation or absence of sexual development usually accompanied by diminished growth. The effect of specific vitamin deficiency on the endocrine glands is profound, although the pituitary gland seems unaffected except for basophil hyperplasia.

There is an interference with adequate absorption from the gastro-intestinal tract caused by chronic diarrhea. This results in a definite lack of vitamins with a consequent effect on growth (vitamins A, D, E and riboflavin), gonadal structure and function (vitamins B1 and E), and cellular metabolism (vitamins B, C and riboflavin). In addition, there may be a deficiency of all the minerals with subsequent osteoporosis from a lack of calcium.

Davidson believes that "In view of the importance of these substances for normal cellular metabolism, it is not surprising that chronic deprivation of them should have considerable effect not only on the organism as a whole but more specifically on the pace makers, the endocrine glands. These organs are so interdependent that a disturbance in one readily affects the other."

The autopsy report in the case described here revealed an ileocolonic fistula and a chronic ulcerative enteritis. The etiology of the fistula is unknown but it may be assumed with some reserve that it was tuberculous in origin. From these changes in the intestinal structure the patient had a resultant marked diarrhea, with other severe gastro-intestinal symptoms. The accompanying lack of vital food materials without a doubt exerted its influence on the endocrines, and from the work of Davidson and others, it is possible to appreciate what could have happened in this patient.

The rational treatment of Simmonds' disease is based on an understanding of the pathologic physiology. A brief reference to the diagram that is included would indicate that the choice method of therapy would be large doses of anterior pituitary extract. Since at autopsy gross and microscopic atrophy of the thyroid, parathyroid, gonads and suprarenals were seen, it seems logical to supplement the pituitary therapy with insulin, cortin, thyroid, and androgenic substances. The use of insulin is not without danger, and in this patient it precipitated a severe hypoglycemic reaction on several occasions.

The patient was treated vigorously under the previously outlined principles. He was given large doses of pituitary extract and appeared to improve with their administration. Particularly noted was a subjective feeling of well-being, an increase of strength, and some disappearance of the gastro-intestinal symptoms. Adrenal cortex extracts were added to his therapy but did not seem to produce any changes worthy of note.

In January, 1939 androgenic substances (testosterone propionate) were given by injection. At that time the patient seemed to undergo a phenomenal change. He became much more cheerful, mental depression disappeared, there was an increase of strength, and a considerable gain of weight. With these injections an increase in size of the external genitalia, growth of pubic hair, and occasional penile erections were noticed. When this treatment was discontinued for several months a reversion to his former distressing symptoms was seen.

The supplemental treatment with thyroid did not seem to aid, either subjectively or objectively, although over a period of four months the basal metabolic rate was elevated from −33 per cent to −10 per cent. However, there was an increased weight loss so this therapy was discontinued and in a short time the basal metabolic rate was depressed to −28 per cent.

The response to anterior pituitary preparations was gratifying and forged another link in the chain of strong presumptive evidence of pituitary dysfunction present in this case. The poor response to adrenal cortical extracts militates against the possibility of this being a case of Addison's disease.

In addition to the therapy mentioned, large doses of vitamin concentrates were given throughout the illness to combat the loss and lack of absorption of these substances as a result of chronic diarrhea.

SUMMARY

A case is presented exhibiting the symptomatology of pituitary cachexia with an anatomically normal pituitary gland revealed at autopsy. The patient was found to have a healed miliary tuberculosis, an ileocolonic fistula, and a chronic ulcerative enteritis. It is believed that the probable combination of nutritional deficiencies and chronic debilitating disease produced symptoms of profound endocrine dysfunction. Therapy with anterior pituitary extract, androgen and vitamins gave favorable but temporary results for a period of years.

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DIETHYLSTILBESTROL* †

A CLINICAL STUDY

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Female sex hormone therapy at its best is confusing to the practitioner. The sources of the natural glandular products are never well defined. Their standardization is only as accurate as it can be made by human frailty and animal end-point reading. It is not to be wondered that the clinician, faced with a disordered female, is bewildered when he reaches into this pharmaceutical grab bag for help. Here he encounters a great array of preparations, often misleading in their nomenclature and no two of which have the same method of standardization. The advent of a new, active synthetic estrogenic substance which is as potent by oral administration as it is when given intramuscularly promises a spectacular advance. The drug—diethylstilbestrol—has been in the authors' hands for investigation for several years. Its efficacy in replacing the natural estrogens is indisputable. It is inexpensive to manufacture and can be prescribed for on the basis of weight of the pure crystalline drug. These assets make its commercial acceptance desirable and it is hoped that it will be on the market reasonably soon. The following report is a brief review of what is known about the drug clinically and a summary of its use in the endocrine clinic of the Department of Gynecology at the University of Maryland. A complete bibliography of the literature concerning stilbestrol is too extensive to include in this paper, but some of the articles which have been considered of unusual interest will be noted. It must be remembered that the problems related to this drug are still controversial and what is accepted today as the reasonable truth tomorrow may be wrong.

The effect of stilbestrol upon the human patient in general can be said to be that of the natural estrogens. There are qualitative differences, particularly from the investigative standpoint, but these are not important to an understanding of the use of the drug.

Stilbestrol produces definite changes in the uterus. The nonpregnant uterus, either in a resting or a castrated state, responds to the drug by endometrial proliferation. Prolonged stimulation may result in actual hyperplasia of the endometrium. Cessation of the treatment causes menstrual bleeding of a typical withdrawal type. If the drug is continued indefinitely bleeding may also occur during its administration from a hyper-

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plastic or proliferative type of endometrium. This condition may make its appearance in from two to several months after the onset of therapy, being preceded in the noncastrated individual by a period of amenorrhea. In the surgical castrate, stilbestrol has been shown by Lackner and Tulsky (13) and others to increase the volume and size of the uterine cavity and to initiate contractions of the organ. Soule and Bortnick (18), in treating a case of primary hypogonadism, noted a definite growth of the primary and secondary sex organs with the assumption of a typically feminine habit and psychic improvement. The authors know of a similar case in which an infantile uterus was converted into an organ of adult size after approximately a year of treatment. Davis and Boynton (6) cite the case of a twenty-two year old white female who had never menstruated and who presented a prepubertal physique. On an average dose of one milligram a day for a year the patient's bodily configuration became decidedly feminine, the genitalia grew to normal adult state, and the breasts increased in size with the development of deep pigmentation of the areolae. Although some of these patients will bleed during the administration of stilbestrol and most of them after its cessation, no specific instances are known in which a previously absent menstrual cycle has been established by the use of the drug. Abarbanel (1) calls attention to some interesting effects of stilbestrol upon the gravid and postpartum uterus. He found that attempts to produce abortion in the first trimester of pregnancy in five cases were fruitless, either by use of the drug alone in total doses of from 100 to 250 mg. or in combination with potent oxytocics. Conversely, in the last trimester of pregnancy oral doses of from 100 to 150 mg. appeared to be an excellent priming agent for the subsequent action of oxytocics in inducing labor. Stilbestrol alone was found to be ineffectual. Evidence was also presented to indicate that the drug exerts a similar priming effect upon the postpartum and postabortive uterus. Connally, Dann, Reese, and Douglass (4), of the University of Maryland, have previously noted this priming effect upon the postpartum uterus. Peel (16) reached similar conclusions after using the drug in fifty-two patients just prior to the expected onset of labor, and reported that it appeared to be of definite value in increasing uterine tone and the efficiency of contractions during labor. In all reports of the use of stilbestrol during pregnancy it has been noted that these women may tolerate large daily doses, as much as 200 to 250 mg., without appreciable side effects.

Accurate observations of the action of stilbestrol upon the human ovary are lacking. Some workers believe that prolonged administration of the drug may result in the suppression of ovulation and may produce permanent damage to the ovary. Karnaky (10) reported on histologic sections of the ovaries of twenty-four patients who had received stilbestrol in doses of from 25 to 1000 mg. over varying periods of time, from twelve hours to thirty days. He writes, "In all (the sections) follicular cysts were obliterated and the ovary became smooth like the ovary in a pregnant woman at term." He also says, "After stilbestrol is discontinued, the ovary begins anew and in many cases the menstrual cycle returns to normal." This statement, while interesting, is of no significance without detailed histologic and clinical reports for support. Shockaert and Ferin (20) reported that Moricard had observed an increase in size of ovarian grafts after administration of several milligrams of the drug.

Striking alterations in the secondary sexual characteristics are produced by stilbestrol, particularly in prepubertal and hypogonadal patients. In this clinic, while treating vulvovaginitis in children cornification of the vaginal mucosa has been noted in all cases. Many patients have developed breast hypertrophy and growth of pubic hair. On one occasion there occurred menstrual-like bleeding for one day after cessation of the drug. In a few patients, interestingly enough, hypertrophy of the clitoris has been noted. It is believed that this latter observation may indicate androgenic potentialities. This would hardly be surprising in view of the close chemical relationship between the estrogens and androgens, plus the fact that it is generally conceded they may have interrelated physiologic activity. These changes occurring in children under the influence of stilbestrol are transient and usually disappear within two to four months after treatment is stopped. The hypogonadal patient, as has been mentioned previously, presents similar developments with the administration of the drug. In these underdeveloped adults the changes appear to be permanent. Slight to moderate breast hypertrophy, usually with increased pigmentation of the areolae, has been noted frequently. The pubic hair increases and becomes feminine in distribution. There may be an increase in the size and volume of the uterus. A neutral or undifferentiated habitus becomes typically feminine. Great improvement in psyche and libido have been reported. Davis (5) writes of the use of the drug in this type of patient, "The daily administration of a small amount provides sufficient estrogenic stimulus to produce physical and sexual maturity of these underdeveloped young women. The changes induced in these patients in a short period of a month or more are truly astounding."

The healthy patient with a normal menstrual cycle exhibits few alterations when treated with stilbestrol, except for amenorrhea, as has been noted. A few patients complain of tingling and fullness of the breasts which is relieved by stopping the drug.

The normal pregnant woman is not influenced by the drug in large quantities and is able to tolerate it well. This is to be expected when the large quantities of estrogens naturally produced and excreted by these patients during gestation are considered.

The castrated or postmenopausal patient who is given stilbestrol likewise manifests few objective changes although she may experience complete subjective relief. In some, cornification of an atrophic vaginal mucosa may be noted; others will complain of breast discomfort.

With regard to the effects of the drug upon other endocrine organs it may be stated that the anterior hypophysis alone is appreciably influenced by its use. Other glands of internal secretion do not appear to be affected although much work remains to be done upon this phase of its activity. Stilbestrol appears to act upon the anterior lobe of the pituitary gland in the same manner as the natural estrogens by suppressing the gonadotropic activities. This mechanism is supposedly the theoretical basis for relief of the symptoms of the menopause. The anterior hypophysis of the menopausal patient is no longer under the modifying influence of the patient's ovarian hormones, consequently there is an apparent increase in gonadotropic activity of the gland. White and Hunt (19) have shown a similar mechanism concerning diabetes in pregnancy. The use of stilbestrol, and other estrogens together with oral progesterone, results in improvement of the disease and reduction of the usual morbidity associated with this condition during gestation. Its medium of action may be by inhibition of the pituitary diabetogenic hormone. In the future the drug may prove efficacious in treating postmenopausal diabetics, as have the natural estrogens in the hands of Cantilo (2) and other workers. Another effect believed to be produced by way of the hypophysis, by suppression of the lactogenic hormone, is the prevention of breast engorgement and inhibition of lactation in the immediate postpartum patient. Soule and Bortnick (18) have found an excellent response early in the puerperium but an indifferent one later. Diddle and Keetel (7), in treating 100 postpartum women with a dose of 10 mg. a day for the first five days found breast engorgement entirely prevented and lactation delayed but not totally inhibited. Other workers, using larger doses, claim to be able to completely inhibit lactation at its onset and also to check milk production once it has developed, in the majority of patients treated. Connally, Dann, Reese and Douglass (4) reported 70.5 per cent suppression of lactation in 200 patients treated with a dose of 5 mg. per day throughout the postpartum hospital perfod.

Stilbestrol also affects the male generative organs. It has been shown repeatedly that any estrogenic substance will produce definite and often permanent damage to the testes. That this is true of the synthetic drug is well substantiated in a report by Dunn (8). He presented a male sexual criminal with large gonads and an inexhaustible libido who volunteered for experimentation. This patient was given a total dose of 480 mg. of stilbestrol. There was noted an appreciable decrease in size of the testes. Biopsy, by testicular puncture, showed degeneration and atrophy of the tubules, with an absence of spermatogenesis. The patient also developed gynecomastia. The testicular damage seemed to be permanent.

The literature has been filled with reports of the so-called toxicity of this drug. Morrell (15), who has surveyed 257 papers for abstracts, reports variations from one extreme to the other, some being as high as 60 per cent of the patients treated. These reports are misleading. Many of the symptoms listed as being evidence of toxicity are subjective symptoms common to all menopausal patients, regardless of therapy. It has, however, become increasingly manifest that a certain percentage of patients treated with the drug display an idiosyncrasy which shows itself by nausea and vomiting. This idiosyncrasy seems to be central or general in origin rather than local, as it is produced by intramuscular injection as well as by oral use. It can be ameliorated somewhat by the use of enteric coated tablets and by taking the drug after meals. While many authors have found this action distressing, others, including the members of the endocrine clinic of the University of Maryland have found it of little importance. Simple reduction of dosage until the patient has established tolerance has been satisfactory in overcoming this ill effect. The studies of Selye (17) indicate that detoxification of the estrogens, including stilbestrol, occurs in the liver when the drugs are given in overdosage. Karnaky (11) has employed tremendous doses without significant alteration in the blood picture or in biochemical reactions of the blood. Freed, Rosenbaum and Soskin (9), and Koenig and Gustavson (12), along with many others have shown the liver function tests of patients receiving the drug to be normal. Experimental animal work has required tremendous doses of stilbestrol, far exceeding those used by the most radical therapists, to produce changes in the blood picture, liver and kidneys. These changes, when so produced, have in no ways been different from those produced by equivalent amounts of the natural estrogens. Davis (5) administered the drug in large doses to three patients with terminal malignancies and at autopsy noted no changes in the liver, kidneys or adrenals which could be attributed to its use.

Aside from the idiosyncrasy of nausea and vomiting there is one other disturbing side effect, the production of uterine bleeding in the postmenopausal or castrated patient with uterus intact. Its reported incidence varies from 10 to 40 per cent or more. It may occur during the administration of the drug or after its cessation. Natural estrogens often produce similar bleeding. This is no cause for alarm but merely calls for reduction of the dose or temporary discontinuance of the drug. Undue alarm on the part of the patient should be prevented by instruction as to the possibility of its occurrence.

The consensus of opinion as to the dose of stilbestrol is fairly uniform. The optimum dose is from $\frac{1}{2}$ to 1 mg. per day. Larger doses are seldom indicated, except during gestation, and when used will decidedly increase the incidence of nausea and vomiting. In the menopausal patient an attempt should always be made to reduce progressively the dose after a satisfactory response has been elicited. This has been successful here and the majority of patients have been able to do without the drug once they have had a satisfactory course of treatment. Those patients who continue to need the drug may be maintained successfully upon as little as $\frac{1}{10}$ mg. two or three times weekly.

Stilbestrol has been most satisfactory in the treatment of the menopause, both natural and operative. It has also been highly effective in vulvovaginitis in children. In general, whenever used it has been more satisfactory to the patient than any of the natural estrogens. Experience with its use in treating dysmenorrhea, while limited, has been gratifying. Other workers have demonstrated its apparent value in hypogonadism, diabetes in pregnancy, suppression of lactation, priming action on the gravid and postpartum uterus, senile vaginitis, senile vulvitis, and pruritis vulvae. Its action in other endocrine problems such as functional bleeding, sterility, loss of libido, and postmenopausal diabetes has yet to be investigated fully. Its effectiveness in arthritis of the menopause and postmenopausal psychoses is distinctly disappointing. The drug may be administered by oral tablet, preferably enteric coated and on a full stomach, by intramuscular injection, by inunction, and by suppository.

In this clinic a total of 115 cases has been treated. Thirty-eight of these were menopausal, twelve operative, and twenty-four natural. Of these thirty-eight patients, the response to therapy was excellent in thirty-three, fair in two, and poor in three. The three who had a poor response also had arthritis and psychoneurotic tendencies, so that evaluation of their symptoms was difficult. None of these three responded to natural estrogens either. The most dramatic relief of symptoms was shown by six of the patients with an artificial menopause. All were young women in whom the subjective symptoms, particularly hot flushes, were so disturbing as to make them chronic invalids. None was relieved appreciably by natural estrogens. All six have been completely asymptomatic with an initial dose of 1 mg. a day and eventually an average maintenance dose of $\frac{2}{10}$ to $\frac{3}{10}$ mg. per day. This small amount is still necessary as there is a recurrence of symptoms with its cessation.

Seven patients with amenorrhea and oligomenorrhea have been treated for short periods of time with no response.

Oral stilbestrol was administered to six patients with senile vaginitis. One responded well, the others had no relief. None of these, however, was given the drug in suppository form.

The drug was given to eight patients with primary dysmenorrhea. Five of these had complete relief of symptoms; the three failures did not respond to repeated courses. Two of the five who experienced relief had a moderate recurrence of symptoms after two normal periods. An additional course of the drug was successful and has remained so to date. The procedure is to administer 1 mg. a day for fourteen days beginning with the first day of menstruation and then wait until the next period has elapsed.

Four patients with functional uterine bleeding were treated with from 2 to 5 mg. a day until bleeding stopped, then were given 1 mg. a day for another seven days. Two patients exhibited an apparent good result but it is questionable whether the drug was responsible.

Four patients with loss of libido were treated with 1 mg. a day. Two were functional in origin and were unrelieved; the other two, as a result of radium therapy, responded excellently.

One patient who had a complete mastectomy and plastic repair for virginal hypertrophy of the breasts, with the subsequent development of oligomenorrhea and hot flushes, was treated with an average of $\frac{1}{2}$ mg. a day. Normal menses were reestablished and the flushes were relieved.

Forty-seven cases of gonococcal vulvovaginitis in children have been treated, with forty-two cures and five recurrences. These have been discussed fully in a previous paper (3).

Nausea and vomiting were noted in 6.3 per cent of all cases. In those patients receiving 1 mg. a day or less the incidence was 4.1 per cent. Uterine bleeding caused by the drug, excluding patients with artificial menopause and functional bleeding, was noted in 13.9 per cent of the total. The children proved the most tolerant group. Of the forty-seven, only one developed nausea on 1 mg. a day. Several tolerated 3 mg. a day without untoward reaction. Only one child exhibited uterine bleeding at the end of the course and bled for one day. The adults developing nausea and vomiting responded well to a reduction of dosage until tolerance was established. Approximately 10 per cent of the patients have taken the drug for six months or more; none of these has shown any cumulative effects.

Seventeen unselected patients who had received or were taking the drug were given a bromsulphthalein liver function test. The basis of the test was 5 mg. of bromsulphthalein per kilo of body weight, as originally suggested by Magath at Rochester in 1935 (14). The normal reading was interpreted as 6 per cent retention or less at the end of one hour. The tests were normal in all seventeen patients. Eight of them complained of an artificial menopause, the doses they received varying from a total of 38 to 273 mg. Seven patients had a natural menopause; their total doses varied from 47 to 273 mg. One patient presented functional bleeding; her dose was 40 mg. The seventeenth patient complained of virginal hypertrophy of the breasts with a postmastectomy menopauselike syndrome. She received a total dose of 73 mg.

Three of the above cases are of sufficient interest to be reported in detail, two because of reactions to antiluetic therapy, the other because of a biweekly injection of bichloride of mercury for lichen planus over a period of two years.

CASE REPORTS

Case No. 1: A 37 year old female negro reported natural menopause with severe symptoms that did not respond to sedatives. She was given 1 mg. a day for 119 days, then 2 mg. daily for eight days prior to the test. The total dose was 135 mg. During the fourth month of treatment with stilbestrol this patient developed a moderately severe reaction to antiluetic therapy, with stomatitis and a transient jaundice. This subsided three weeks before the bromsulphthalein test was made. The patient had an excellent response to therapy; there was no nausea or vomiting.

Case No. 2: The patient was a 31 year old white female. She complained of artificial menopause with severe symptoms which did not respond to sedatives and responded only fairly well to natural estrogens in large doses. The dose prescribed was 1 mg. daily for 126 days, a total of 126 mg. The response was excellent; there was no nausea or vomiting. Two years previously this patient had had a severe attack of postarsphenamine jaundice for which she was hospitalized. One year following this she developed purpura which was thought to be the result of continued antiluetic treatment. The luetic therapy was discontinued and the purpura cleared up. At the time of the liver function test the serologic test for syphilis was positive.

Case No. 3: This was a 34 year old white female patient who reported artificial menopause with moderately severe symptoms that did not respond to sedatives. She was given 1 mg. of stilbestrol daily for forty-two days, or a total dose of 42 mg. The response was excellent; there was no nausea or vomiting. The patient had received a biweekly intramuscular injection of 1/12 gr. of bichloride of mercury for lichen planus over a period of two years prior to stilbestrol therapy.

All three of these patients had normal liver function tests.

CONCLUSIONS

Diethylstilbestrol is an excellent, potent, inexpensive, synthetic estrogenic substance which is effective orally. In this clinic it is satisfactorily replacing the intramuscular use of natural estrogens. That a small percentage of patients exhibit an idiosyncrasy to the drug, manifested by nausea and vomiting, is no contraindication to its use. Such an idiosyncrasy can be overcome satisfactorily by a reduction of dosage until tolerance is established. Real evidence of toxicity produced by this drug in the female patient is yet to be proved. The drug is not to be given to male patients, except for the express purpose of producing testicular degeneration and atrophy. The authors, together with many of their patients, express a fervent desire that this useful drug will soon be put upon the market for, as has often been stated in the clinic, "those pills are wonderful, doctor, there's nothing else like them."

The authors wish to extend sincere thanks to Eli Lilly and Company for their generous assistance in this work.

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PARALDEHYDE AS A FACTOR IN LOWERING MAJOR OPERATIVE INTERFERENCE* †

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In reviewing the literature on analgesia it was surprising to find very little concerning its effect upon the incidence in operative obstetrics. In 1930 Gwathmey (8) reported the results of 20,000 cases that received rectal instillations of quinine, alcohol, ether and olive oil, together with magnesium sulfate and morphine hypodermically. After stressing the favorable amnesic and analgesic effects in this series he summarized its influence on delivery by the following conclusions: 1. The progress of labor is not delayed. 2. Occiput posterior positions rotate in about the same proportion as normal cases. 3. Postpartum hemorrhage is less than with inhalation methods. 4. Morbidity of the mother is decreased. 5. No fetal deaths are attributed to analgesia. 6. There is less postpartum fatigue.

In 1932 Rosenfield and Davidoff (17) first reported the use of paraldehyde in a series of fifty cases. Colvin and Bartholomew (3) in 1935 compared their results in using this same drug with Rosenfield's cases, and with those of Irving (10) who used the Gwathmey method, and concluded that operative interference was not increased as compared to patients receiving no analgesia.

Other reports have not been so enthusiastic. In studying 108 anesthetic deaths occurring in Philadelphia from 1931 to 1935, Montgomery (15, 16) made this statement concerning analgesia. "If the patient is in constant danger of injuring or contaminating herself, if her cooperation in the course of labor is utterly lost, *if the incidence of operative intervention is multiplied tenfold*, if supervision of the case is transformed from the intelligent conduct of labor into treatment of drug confusion, one doubts the effect is worth the reaction that is produces." Many other authors are of the same opinion, so with this in mind it was thought that it would be of interest to see the effect paraldehyde has on the incidence of operative interference since its introduction as a routine analgesic at the University Hospital.

A series of 200 cases which received morphia and pentobarbital, or no analgesia, was compared to a similar number receiving pentobarbital and paraldehyde. Both groups were delivered or supervised by the same staff and there was an equal number of service and private patients. Any case in which it was thought that analgesia played no part in the course of labor

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was omitted. This included many patients in whom the delivery was terminated without regard to the labor, such as hemorrhage or cardiac disease complicating pregnancy, transverse presentation, disproportion, or elective cesarean section.

The course of therapy was similar to that reported by Douglass and Peyton (6). In primigravidas when the cervix had dilated to 5 cm. and pains were strong and regular, 3 gr. of pentobarbital was given and followed in fifteen minutes to one-half hour by the oral administration of 6 dr. of paraldehyde mixed with an equal amount of aromatic elixir. If this was vomited a similar dose was repeated in fifteen minutes. The effects of the paraldehyde lasted from three to six hours and it was repeated in doses ranging from 3 to 6 dr. when the patient showed signs of the drug diminishing in its effect. In multigravidas the pentobarbital and paraldehyde were given when the pains occurred every two to three minutes and lasted forty-five to sixty seconds, regardless of cervical dilatation.

TABLE 1

	(200) WITHOUT PARALDEHYDE					PARALDEHYDE (200)				
400 patients	Private multip.	Service multip.	Private primip.	Service primip.	Total	Private multip.	Service multip.	Private primip.	Service primip.	Total
	per cent	*per cent*	*per cent*	*per cent*	*per cent*	*per cent*	*per cent*	*per cent*	*per cent*	*per cent*
Occiput anterior	92	80	72	86	82.5	82	84	72	68	76.5
Occiput transverse	0	4	8	0	3.0	6	2	12	20	10.0
Occiput posterior	0	12	20	10	10.5	10	12	14	10	11.5
Breech presentation	8	4	0	8	5.0	2	2	2	2	2.0
Low forceps	16	18	36	36	26.5	62	54	90	92	74.5
Midforceps	4	12	24	6	11.5	2	0	8	4	3.5
Spontaneous delivery	72	66	40	50	57.0	34	42	0	4	20.0
Duration of labor (hours)	9.3	16.8	17.7	19.6	15.8*	6.8	12.8	18.6	16.2	13.6*
Maternal morbidity	12	12	12	12	12.0	6	10	10	20	11.5
Fetal mortality	4	8	4	8	6.0	4	6	2	2	3.5

* Average.

The first four lines in table 1 show the similarity between the two series. The slight increase in the number of occiput transverse presentations in the paraldehyde group probably was caused by the use of control forceps when the head reached the perineal floor, but before spontaneous rotation occurred. The use of low forceps has increased from 26.5 per cent to 74.5 per cent.

The routine use of control forceps was first advocated by DeLee (5) and since then has been adopted by a great many obstetric clinics. It is not considered a major operation and, when combined with an episiotomy, very little traction is necessary and allows a slow controlled delivery of the head, avoiding the hazards to the infant's brain of a prolonged perineal delivery.

The application of midforceps has decreased from 11.5 per cent in the first series to 3.5 per cent in the paraldehyde group. The indications for delivery when the presenting part is in the midpelvis have decreased most in the treatment of private primigravidas and service multiparas. Here analgesia plays one of its most important roles. The fact that the private patient is unable to complain while in labor has enabled the obstetrician to follow a course of watchful waiting, allowing labor to progress and terminate by a simpler delivery. Both the mother and the child benefit from this procedure. Cole in a recent article (2) reported that any factors which tend to retard or ease the second stage of labor seem to be favorable for the child. In the handling of service multiparas it should be explained that the majority admitted to the University Hospital presented some type of complication. Many of these were unregistered patients who had been neglected during the first stage of labor. Here again, paraldehyde with intravenous fluids and similar measures have enabled the clinic to practically eliminate the use of midforceps.

Contrary to popular opinion, the duration of labor was less in the paraldehyde group. If the routine use of control forceps, which were used approximately two hours before the patient would have delivered spontaneously, were taken into consideration, the duration of labor would be about the same in the two groups. Although uterine contractions do not occur as frequently in the patients receiving paraldehyde, they apparently are more effective because there is less fatigue.

Van Hoosen (19), Montgomery (15, 16), and others feel that the maternal morbidity is greatly increased when analgesia is used. It is admitted that patients are restless and frequently touch the genitalia with their hands, but from the study made at the University Hospital the rate of infection is the same in both groups. Evidently these patients have built up an immunity against their own germs and as long as a new strain is not introduced the morbidity rate is not increased.

The fetal mortality rate has been reduced 45 per cent. Kotz and Kaufman (14), and Schrieber (18) have shown that infants under the influence of paraldehyde do not cry as readily as other babies, but respirations do occur early and if they are not resuscitated too vigorously, but are placed in a warm crib with their heads lowered, the end results are excellent. DeLee (5) observed that infants also need analgesia during labor. He noted that when the fingers were pinched in utero the infant withdrew its hand.

Since one cannot draw too many conclusions from such a small series, table 2 was compiled. This shows all the deliveries that occurred at the University Hospital four years before and four years after paraldehyde was introduced as a routine procedure. It represents a total of 5800 hospital deliveries. Of the latter group (1936 to 1939 inclusive) approximately 66 per cent received paraldehyde. The remainder included patients in whom analgesia played no part in the course of the delivery.

An analysis of the indications for the use of high and midforceps revealed that the most common cause for the termination of labor was maternal and fetal distress, or both. This was given as the reason for termination in ten of the thirteen cases of high forceps. In the midforceps group, distress associated with prolonged labor, occiput posterior and occiput transverse positions comprised 86 per cent of all the indications for the entire eight years. Fetal and maternal distress associated with prolonged labor alone were the indications in 43 per cent of the group receiving very little analgesia and in only 20 per cent in the series in which paraldehyde was used. In comparing the total number of patients delivered with the number of indications for midforceps, fetal and maternal distress accompanied by prolonged labor occurred in fifty-six of 1932 patients (1932 to 1935), and in only twenty-two of 3880 deliveries (1936 to 1939), or an 80 per cent reduction. Occiput posterior and occiput transverse positions were reduced 40 per cent and 10 per cent respectfully. The maternal morbidity and fetal mortality rates compare favorably with the data of table 1.

TABLE 2

	NORMAL DELIVERY	HIGH FORCEPS	MID FORCEPS	LOW FORCEPS	VERSION AND BREECH EXTRACTION	BREECH EXTRACTION	CESAREAN SECTION	FETAL MORTALITY	MATERNAL MORTALITY
	per cent	*per cent*	*per cent*	*per cent*	*per cent*	*per cent*	*per cent*	*per cent*	*per cent*
1932–1935 (incl.).....	48.8	0.8	6.7	24.5	2.6	5.9	10.7	11.5	2.1
1936–1939 (incl.).....	29.8	0	3.0	56.5	0.7	4.2	5.9	8.9	0.7

From this study the most striking feature noted was the effect that paraldehyde has had on the course of labor. This effect is accredited directly to the lowering of the incidence of maternal and fetal distress. Formerly these cases required major forceps operations to be delivered. One might say that this improvement is the result of a more conservative attitude in the handling of difficulties that arise in the practice of obstetrics. This is true; however, paraldehyde has played a major part in allowing such a course to be followed.

SUMMARY

1. Two hundred patients who received little or no analgesia are compared with an equal number receiving pentobarbital and paraldehyde. All were delivered or supervised by the same group of men.

2. The incidence of occiput transverse presentation is greater in the analgesic group because of the routine use of control forceps.

3. The indications for midforceps are decreased from 11.5 per cent to 3.5 per cent.

4. The fetal mortality and maternal morbidity are the same or less in the paraldehyde series.

5. The duration of labor averages two hours less in the analgesic group.

6. A comparison of all the operative deliveries occurring four years before and after the introduction of paraldehyde shows that fetal and maternal distress, associated with prolonged labor as an indication for major forceps deliveries, are reduced 80 per cent.

7. Analgesia plays a major part in allowing the clinic to practice a more conservative role in the treatment of obstetric difficulties.

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THE DECREASE IN THE INCIDENCE OF THERAPEUTIC ABORTIONS

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Within the past few years there has arisen throughout the country a growing consciousness of maternal welfare. This trend is evidenced by the appearance of numerous active maternal mortality committees. So also should the physician look upon the falling incidence of therapeutic abortion as another index of the progress being made in maternal care.

With this in mind the authors have collected and attempted to analyze a series of therapeutic abortions performed in the University Hospital over a period of thirteen years. An effort has also been made to discuss briefly the more recent concepts on this subject. Data have been compiled which show a decided reduction in the number of therapeutic abortions performed and indicate how this progress has been made.

As may be seen in table 1, the cases have been grouped into three series according to the date of admission in order to facilitate better comparison. Thus, in the first series from January 1, 1924 until December 31, 1931 there were 2880 obstetric patients admitted to the hospital, twenty-three or 0.8 per cent of whom were therapeutically aborted. The second and third series showed a decrease in the percentage of therapeutic abortions performed of 0.43 per cent and 0.29 per cent, respectively. In more graphic terms, the incidence of therapeutic abortions has been reduced 63.7 per cent from the first to the third series.

Table 2 shows the indications for which therapeutic abortions were performed at the University Hospital, together with the number performed in each series. Briefly, the indications for therapeutic abortions were grouped into three or four classes. The first group included the toxemias and related conditions of pregnancy including hyperemesis, renal disease, pyelitis, hypertensive disease, preeclampsia and eclampsia. The last two mentioned rarely occurred prior to the last trimester and hence were infrequent indications for therapeutic abortion. The second and third groups, respectively, were heart disease and tuberculosis. Stander in his recent textbook also gave a fourth group of indications composed of severe diabetes and such rare diseases as leukemia, pernicious anemia, chorea, and, seldom, mental diseases. Interestingly enough, there were no indications

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for these conditions found among the cases cited in the series at the University Hospital.

A study of table 2 shows that in the second and third series the reduction in the incidence of therapeutic abortion has resulted almost entirely from the improvement in the treatment of hyperemesis. Lesser significance may be attached to the reduction in the number aborted for heart disease, tuberculosis, and pyelitis. The appearance of preeclampsia, hypertensive disease, and posteclampsia in the last series may be attributed to the more recent classification of toxemias. There has been no improvement in the statistics of therapeutic abortions for renal disease, hypertensive disease, preeclampsia or eclampsia. These make up a group of conditions about which there is much to be learned, and although the pathology has been considered thoroughly the treatment is largely empirical and all too frequently inadequate. As yet there has been found no cure for chronic nephritis, and the best treatment for preeclampsia and eclampsia is prevention.

TABLE 1

	NO. OF PATIENTS ADMITTED TO THE HOSPITAL	THERAPEUTIC ABORTIONS	PERCENTAGE ABORTED
1-1-24 to 12-31-31	2880	23	0.80
1-1-36 to 6-30-38	2520	11	0.43
7-1-38 to 12-31-40	3687	11	0.29

TABLE 2

Indications

	FIRST SERIES	SECOND SERIES	THIRD SERIES
Hyperemesis	11	5	0
Renal disease	4	5	6
Hypertensive disease	0	0	1
Preeclampsia	0	0	1
Posteclampsia	0	0	1
Tuberculosis, pulmonary	3	1	1
Heart disease	1	0	0
Hemorrhage	1	0	1
Tuberculosis and hyperemesis	1	0	0
Pyelitis	1	0	0

In explanation of table 2 it should be mentioned that the two patients aborted for hemorrhage were both cases of hypertensive disease in which a complete premature separation of the placenta occurred during the twenty-sixth and twenty-seventh weeks of pregnancy. These might better be classified as the completion of inevitable abortions. By *posteclampsia* is meant that period in the disease when the convulsions have been controlled but the toxemia is still present in a more or less alarming degree.

As may be seen, the incidence of therapeutic abortion in patients with tuberculosis has decreased from three cases in 2880, or 0.1 per cent, to two out of 6200 admissions, or 0.03 per cent. This incidence appears to be in keeping with the recent literature. Bridgman and Norwood found in a series of pregnant patients with tuberculosis that the mortality was lower in the group of patients allowed to carry the pregnancy to term under proper care than in the group aborted.

Schaefer and Law in the *Quarterly Bulletin of Sea View Hospital* recently reported a series of 231 full term pregnancies complicated by tuberculosis, together with thirty-five induced abortions for the condition. The cases were grouped according to the severity of the lesions and the results compared with a control series of nonpregnant patients having similar pulmonary lesions. The classification used was caseous pneumonic, chronic productive, exudative productive, and arrested tuberculosis. Meticulous medical and obstetric care were stressed in the handling of these patients and the method of delivery was shown to depend upon the type of pulmonary involvement. The authors concluded that the figures indicated that pregnancy usually had no ill effect on pulmonary tuberculosis, provided both the pregnancy and the tuberculosis were managed carefully. However, they felt that therapeutic abortion was indicated definitely in two circumstances in the first trimester: First, for all cases of far advanced bilateral caseous pneumonic tuberculosis and, second, where the pulmonary lesion had not been arrested for at least two years.

Thus it appears that here, too, is an indication that in many clinics the treatment now is much more conservative. The two patients aborted for tuberculosis at the University Hospital during the past five years were both private ones. In this clinic the interruption of pregnancy for pulmonary tuberculosis is becoming a rare procedure and one to which much thought is given.

The problem of heart disease in pregnancy is a broad one in which the question of interruption, particularly among the internists, has played an important role. Here again it is the policy of the clinic to be conservative, as pointed out by the fact that only one patient was aborted for this indication in the first series and none during the past five years.

From Jensen's book *The Heart in Pregnancy* one receives the impression that he felt an exaggerated notion of the seriousness of the lesion all too frequently led to radical treatment in the pregnant woman. In a discussion of therapeutic abortion in relation to rheumatic cardiovascular disease, Jensen reported that there was but one cardinal indication for interruption and that was cardiac failure that did not respond to treatment. According to him, mitral stenosis, auricular fibrillation, myocarditis, and a history of previous decompensation, though rendering the prognosis less favorable, were not indications for termination of a pregnancy. As for the relation of therapeutic abortion in other cardiac conditions, he said little owing to the infrequency of their occurrence. He did however, mention the congenital heart and thought that if the disease was compatible with adult life, it rarely interfered with pregnancy. Stress should be placed on prenatal care, for, as in other problems, early recognition and treatment should reduce materially the incidence of maternal and fetal calamities.

From table 2 it is evident that the decrease in abortions for hyperemesis was the most outstanding. In view of the marked success in the treatment of hyperemesis a group of 114 consecutive cases appearing throughout this series of abortions is reported.

TABLE 3

Hyperemesis

	FIRST SERIES	SECOND SERIES	THIRD SERIES
Number of cases	43	32	39
Percentage of hospital admissions	1.49	1.27	1.05
Therapeutic abortions	11	5	0
Maternal deaths	3	0	0
Abortions in patients receiving prenatal care	?	1	0
Abortions in patients not receiving prenatal care	?	4	0

All patients admitted were those who did not respond to treatment through the prenatal clinic, or who when first seen were too ill to attempt treatment outside of a hospital. The percentage of hospital admissions for hyperemesis showed a slight decrease as compared with the succeeding series. This probably was the result of more adequate prenatal care through a better understanding and treatment of the condition. Thus with vitamin therapy and diet it was possible to treat the more ambulatory patients successfully.

In the first and second series of cases the treatment consisted of complete isolation, intravenous glucose, and subcutaneous infusions of salt solution with various types of psychotherapy. A number of patients in the first series was referred to the hospital for therapeutic abortion after inadequate or poor prenatal care. In these cases abortions were performed immediately or shortly after admission, which probably account for the extremely high incidence and the deaths in this group.

All patients in the second series received intensive treatment before it was decided to resort to interruption. It is interesting to note that four of the patients aborted in this series received inadequate or no prenatal care. Five of the thirty-two in this series or 15.6 per cent of the cases were interrupted. This compared favorably with Irving's figure of 17.3 per cent before institution of his new therapy.

The third series included all cases of hyperemesis treated in the hospital from July, 1938 to January, 1941. These patients received the same intensive treatment plus the addition of intravenous or intramuscular injections of thiamin chloride in daily doses of 25 to 75 mg. After about twenty-four to forty-eight hours of such treatment they were placed on frequent small feedings of cereals and pureed vegetables, and the following day were given frequent small amounts of water by mouth. The diet was kept high in vitamin content and gradually increased. Upon being discharged the patients were required to return to the hospital every other day for intramuscular injections of thiamin chloride, and after a week or so were placed on vitamin B_1 by mouth, 5 mg. two to four times a day. The results have been most gratifying, since in this series it has been unnecessary to interrupt a single pregnancy and there have been no maternal deaths.

Two of the five patients therapeutically aborted in the second series returned because of subsequent pregnancies. One of these cases was included in the third series. One patient complained of moderate morning sickness but otherwise had an uneventful prenatal course. The other was referred to the clinic by a private physician because of severe hyperemesis. Her condition appeared worse than during her first pregnancy. On admission the patient was markedly dehydrated, had been vomiting incessantly for two weeks, and was thoroughly convinced that another interruption of pregnancy was the only solution. Her icteric index was 10.6 and the nonprotein nitrogen 52 mg. per cent; the urinalysis showed a trace of albumin. Immediate treatment was instituted, to which the patient responded and was successfully carried to term. It was felt that this resulted almost entirely from careful prenatal care and the use of thiamin chloride.

These data compare favorably with those of Irving reported in the *Virginia Medical Monthly* of December, 1940. Here he discussed at length the etiology, pathology, symptoms, prognosis, and treatment of this condition. He called attention to the apparent success of desensitization with progesterone, and the various hormonal and vitamin therapies. Of chief interest, however, was the remarkable success with which he treated 225 consecutive severe cases. He reported that he was able to reduce the incidence of therapeutic abortion from 17.3 per cent to 2.6 per cent, and the maternal mortality from 5.5 per cent to zero. His mode of therapy was complete isolation, rectal barbiturates, hypodermoclysis of salt solution, and intravenous infusions of glucose, followed later by forced feedings through the use of the Levin nasal tube. This therapy was supplemented by intramuscular injections of ascorbic acid, liver extract, and thiamin chloride. Thus he attributed his success to the combatting of starvation by adequate fluid intake, and the innovation in the use of the Levin tube with large doses of essential vitamins. In conclusion he expressed the opinion that there will always be a few cases in which no treatment will succeed. He felt that if, despite treatment, there was a steadily rising pulse rate remaining well above 110 or if there was still persistent vomiting with jaundice, interruption of pregnancy was indicated.

In summation, it is shown that the incidence of therapeutic abortion at the University Hospital clinic has been reduced 63.7 per cent in the last two and a half years over former statistics. This reduction largely is attributable to a fall in the number of cases aborted for hyperemesis gravidarum. Figures show no therapeutic abortions performed in the past two and a half years for this condition in contrast to a 15.6 per cent incidence in previous years. It is felt that the improvement is effected chiefly through the institution of intensive vitamin therapy. The incidence of abortion for tuberculosis is reduced from 0.1 per cent to 0.03 per cent in the last two series, and no cardiac cases are interrupted in the last 6200 obstetrical admissions to the hospital. The incidence of therapeutic abortion for renal and hypertensive disease remains approximately the same throughout.

In conclusion, too much emphasis cannot be placed upon the early recognition and treatment of complications in pregnancy. This can only be done through proper prenatal care, with an appreciation of obscure symptoms that may be indications of more serious trouble in the future. It should be remembered that each therapeutic abortion is in reality not only an admission of inadequacy on the part of the medical profession for the treatment of certain conditions, but also the deliberate destruction of a potential life. The successful termination of a pregnancy with both a live mother and baby is, in reality, the ideal goal for which one should strive.

FECAL IMPACTION CAUSING SEVERE PAINS IN THE LEGS OF A CHILD*†

A Case Report

CLEWELL HOWELL, B.S., M.D.

TOWSON, MD.

Occasionally a case seems worth reporting not because of the rarity of the condition or the diagnostic acumen needed to solve it, but simply because of the light it sheds on the possible symptoms of a common ailment. Such a case is herewith presented.

CASE REPORT

A. A., a 7 year old white girl, had been having excruciatingly painful cramps in her legs for several days.

The family and past history of the patient were unimportant.

The present illness began five days prior to the time she was seen with the complaint of spasmodic pains from the knees to the ankles. These pains were said to last from a few minutes to an hour, and recurred several times a day. They were so severe that the child would cry out and become pale and limp. Once or twice she complained of being nauseated, yet she did not vomit or mention pains in her stomach. There was no elevation of temperature and between pains she appeared normal. No change was noted in her appetite, and both she and her father (the informant) reported that her bowels had been moving regularly. During the five days before she was seen by the author she had been under the care of her family physician and had been given large quantities of analgesics, such as aspirin and codeine, without apparent effect on the paroxysms of pain.

Upon examination the patient appeared to be small but fairly well nourished. She seemed bright and alert; there was no evidence of toxicity and no fever. Once during the examination she suddenly became pale, perspiration appeared on her forehead, and she cried out with pain in the calves of her legs. In a few moments, however, the pain subsided and she appeared normal. A physical examination was essentially negative, except for a moderate fecal accumulation which could be felt through the abdominal wall. She was hospitalized for further study.

Laboratory studies at the hospital showed 4,430,000 red blood cells; 9,300 white blood cells; hemoglobin 74%; polymorphonuclears 65%; lymphocytes 33%; eosinophils 2%. The urinalysis was normal. Serologic tests for syphilis were negative. The sedimentation rate was normal. An examination of the stool, when obtained, was positive for occult blood.

During the first few hours in the hospital the patient was uncomfortable, and large doses of aspirin, phenobarbital and codeine failed to relieve her discomfort. Her rectal temperature was never higher than 100 F.; the pulse rate ranged from 90 to 120.

As soon as the examinations and laboratory studies were completed she was given a cleansing enema and large quantities of hard fecal matter were passed. Within a half

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* From the Department of Pediatrics, School of Medicine, University of Maryland.

† Received for publication July 15, 1941.

hour after the enema the pains ceased and the patient went to sleep. There was no further recurrence of pain. The next day the child was discharged from the hospital with a diagnosis of fecal impaction and when last seen there had been no return of symptoms.

SUMMARY

A case of fecal impaction is reported which is of interest because of the lack of localizing symptoms and because of the extreme bilateral cramping pains in the calves of the legs caused by this impaction.

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ITEMS

Capt. Frederick L. McDaniels, (U. of Md. 1913) Medical Corps, United States Navy, has been appointed executive officer of the Naval Hospital at Great Lakes, Illinois. Before going to Great Lakes, Captain McDaniels was stationed at the Naval Hospital at Philadelphia. A member of the Navy since April, 1917, Captain McDaniels was on duty with United States destroyers operating in European waters during the first World War. Following the cessation of hostilities in 1918, he saw service in the West Indies, Washington, D. C., and with the Pacific fleet.

Captain McDaniels attended Auburn University, George Washington University, and the Royal College of London in England. After receiving his degree of Doctor of Medicine from the School of Medicine, University of Maryland, he was a resident at the University Hospital and St. Joseph's Hospital in Baltimore, and the Kansas State Hospital, Osawtomi, Kansas. Because of his outstanding work, he was named Fellow of the American College of Physicians and Fellow of the American Psychopathic Association.

A TRIBUTE TO DR. JOHN D. ROBINSON

Through *The Wallace Enterprise*, the newspaper of Wallace, North Carolina, citizens and business concerns paid tribute to Dr. John D. Robinson on his twenty-fifth anniversary as a physician.

Dr. Robinson attended Davidson College and received his degree in medicine from the University of Maryland in 1915. He interned for one year at the James Walker Memorial Hospital in Wilmington and started the practice of medicine in Wallace in 1916. He joined the Medical Corps of the United States Army in 1917 and served overseas for several years. While stationed in Great Britain Dr. Robinson found time to do post-graduate work at the University of Edinburgh. Upon his return to the United States he resumed his practice in Wallace. In 1924 he was commissioned a Major in the Medical Reserves.

The tribute of his friends and associates was most gratifying to Dr. Robinson. Few communities take the opportunity to express their sincere appreciation and deep respect for one who has served them so faithfully and untiringly.

Dr. Thomas B. Aycock, Clinical Professor of Surgery, delivered an address on "Surgery in the Community: Control of Tuberculosis" at the meeting of the American Hospital Association held in Atlantic City on September 16, 1941. Dr. Aycock also spoke on "Surgical Treatment of Duodenal Gastric Ulcer" at the Martinsburg City Hospital, Martinsburg, West Virginia on May 6, 1941, and on the same subject at the meeting of the Augusta County Medical Society held in Staunton, Virginia on May 7, 1941.

Dr. Howard Brooks Mays, Instructor in Genito-Urinary Surgery and Assistant in Pathology, spoke on "Diagnosis and Treatment of Urogenital Tumors" at the September meeting of the Baltimore County Medical Society which was held on September 17, 1941.

Dr. Thomas S. Saunders (U. of Md. 1932) has been appointed consulting dermatologist to the Multnomah County Hospital, Portland, Oregon.

Dr. Timothy A. Callahan, Jr., (U. of Md. 1938) has been commissioned a Lieutenant in the British Army and left for England in July to join the British Army Medical Corps.

Dr. Leo Hochfeld (U. of Md. 1937) has opened an office at 1240 Walton Avenue, New York City, for the practice of medicine and surgery.

Dr. J. Earl Kadan (U. of Md. 1937) announces the opening of an office in the Farragut Medical Building, Washington, D. C. His practice will be limited to obstetrics and gynecology.

Dr. Jack H. Woodrow (U. of Md. 1937) has opened his office at 139 Locust Hill Avenue, Yonkers, N. Y. for the practice of diseases of the eye, ear, nose and throat. Dr. Woodrow was recently made a Diplomat of the American Board of Otolaryngology.

Dr. Irving Freeman (U. of Md. 1935) has been commissioned First Lieutenant in the Army Medical Corps and will be stationed at Fort Belvoir, Virginia.

OBITUARIES

Abshire, Joseph T., Kaplan, La.; P. & S., class of 1889; aged 72; died June 22, 1941, of heart block, heart disease and arteriosclerosis.

Bannister, John H., Charleston, W. Va.; P. & S., class of 1912; aged 54; died, June 2, 1941, of bronchopneumonia and trauma to the chest following an automobile accident.

Bay, James Hugh, Havre de Grace, Md.; class of 1908; aged 59; died, July 6, 1941, after an illness of several months.

Beason, William A., Asheville, Ala.; P. & S., class of 1893; aged 73; died, May 23, 1941, of cerebral hemorrhage.

Boland, Daniel Jerome, Worcester, Mass.; B.M.C., class of 1904; served during the World War; aged 62; died, May 27, 1941, of pulmonary infarct.

Braman, Harry Scott, Homer, N. Y.; P. & S., class of 1892; aged 68; died, April 23, 1941, of cerebral thrombosis.

Carman, John Henry, Plainfield, N. J.; P. & S., class of 1881; aged 83; died, July 4, 1941, in Rockland, Maine, of arteriosclerosis and heart disease.

Chrystal, Michael Henry, Leominster, Mass.; B.M.C., class of 1901; aged 72; died, July 13, 1941, of cerebral hemorrhage.

Craig, James Alexander, Gary, Ind.; class of 1908; served during the World War; aged 61; died, July 2, 1941, of mitral regurgitation.

Foley, David Oswald, Mount Jackson, Va.; class of 1896; aged 69; died, April 24, 1941, of angina pectoris.

Freed, Arnold U., Baltimore, Md.; class of 1939; aged 27; died, July 3, 1941, of accidental drowning.

Getz, Charles, Baltimore, Md.; class of 1879; aged 85; died, September 6, 1941, after a brief illness.

Haines, John F., Monroeton, Pa.; P. & S., class of 1888; aged 77; died, July 14, 1941, of cerebral hemorrhage, hypertension and arteriosclerosis.

Heck, John J., Baltimore, Md.; P. & S., class of 1903; aged 61; died, May 4, 1941, of arteriosclerosis and heart disease.

Jamison, Jesse A., Fairmont, W. Va.; P. & S., class of 1884; aged 81; died, May 26, 1941, of coronary heart disease.

Jeffrey, Hugh R., Morristown, Ohio; P. & S., class of 1893; aged 72; died, June 10, 1941.

John, Benjamin Clifford, Morgantown, W. Va.; P. & S., class of 1920; aged 44; died, April 25, 1941, of pulmonary abscess.

Kernan, Charles K., Pulaski, Va.; class of 1887; aged 79; died, April 18, 1941, of arteriosclerosis and pulmonary tuberculosis.

Lautenbach, George W., Baltimore, Md.; class of 1897; aged 64; died, April 9, 1940.

Lilly, Percy Edwin, Kilmarnock, Va.; class of 1901; aged 63; died, June 15, 1941, of angina pectoris.

MacIsaac, John L., Antigonish, Nova Scotia; B.M.C., class of 1907; aged 71; died, March 25, 1941, after a brief illness.

MacPherson, Alexander G., Grand Rapids, Mich.; P. & S., class of 1903; aged 60; died, March 14, 1941, after a long illness.

McGoogan, Benjamin J., Morven, N. C.; class of 1912; aged 52; died, May 22, 1941, of coronary thrombosis.

Mulholland, Bernard Joseph, Lawrence, Mass.; B.M.C., class of 1906; aged 57; died, April 16, 1941, of cirrhosis of the liver.

Patterson, Herbert J., Portland, Me.; P. & S., class of 1885; aged 79; died, May 13, 1941, of carcinoma of the pancreas and biliary calculi.

Purney, John, New Britain, Conn.; B.M.C., class of 1906; served with the Canadian Army during the World War; aged 62; died, July 18, 1941, of coronary thrombosis while on his vacation in Shelburne, Nova Scotia.

Reckard, Hiram Leslie, Baltimore, Md.; B.M.C., class of 1895; aged 69; died, July 27, 1941, of uremia after an illness of eleven weeks.

Silverstein, William Rice, Bradley, N. J.; B.M.C., class of 1903; aged 58; died, June 21, 1941, in the Beth Israel Hospital, Newark, of cholelithiasis.

Smith, C. Franklin, Uniontown, Pa.; class of 1923; aged 44; died suddenly on August 14, 1941, while on vacation at Tilghman Island on the Chesapeake Bay.

Telfair, William Grimes, Orange, N. J.; class of 1882; aged 83; died, July 14, 1941, of carcinoma of the sigmoid and chronic endocarditis.

Walsh, James Henry, Fall River, Mass.; P. & S., class of 1910; aged 56; died, May 14, 1941, of heart disease and hypertension.

Warner, Anthony Kimmel, Chicago, Ill.; class of 1885; aged 77; died, June 27, 1941, of cerebral hemorrhage and arteriosclerosis.

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FUNCTIONAL UTERINE BLEEDING*†

EMIL NOVAK, M.D., D.Sc.
BALTIMORE, MD.

The fact that uterine bleeding is so often a symptom of definite structural pathology in some portion of the genital apparatus, and not infrequently of cancer itself, gives it a tangibility which is not usually shared by the opposite quantitative menstrual disorder, amenorrhea. The latter is far more frequently encountered in women whose pelvic organs are entirely normal, and the cause of the disorder must commonly be sought for in the still foggy field of reproductive endocrinology. And yet in recent years the medical professional has come to realize that menstrual excess, like deficiency or absence of the flow, is also frequently caused by a disturbance of function rather than structure, and these cases of functional bleeding form a very numerous group indeed.

It is to the consideration of these facts that this paper is chiefly directed, because of their importance and the present day interest in the problems of endocrinology. Certainly they constitute a less deadly group than do those cases of bleeding associated with such grave structural lesions as cancer. Unlike the latter there is no case of functional bleeding which cannot be cured if one wishes to resort to sufficiently radical measures, such as removal of the uterus, removal of the ovaries, or abolition of their function. These measures, however, are unthinkable in a large proportion of cases, in which the problem is to effect a cure without impairment of the future functional capacity of the pelvic organs with regard to menstruation and childbearing.

It is this combination of desiderata which makes the therapeutic problem such a difficult one, especially since knowledge of the mechanism of many cases is still so woefully incomplete. For that matter there is yet much that is not known regarding the mechanism of normal menstruation. The latter is often cited as the only example of a purely physiologic type of bleeding, although this is not strictly true. The conspicuous external manifestation of the menstrual cycle is the bleeding.

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* From the Department of Obstetrics, School of Medicine, University of Maryland.

† Read at the Annual Meeting of the Interstate Postgraduate Assembly, Minneapolis, October 13, 1941.

Some believe this flow to be explainable entirely by the estrogen deprivation of the endometrium, some by the withdrawal of progesterone, and others by the presence of an extra-ovarian bleeding factor of a source as yet unknown. The recent studies of Markee indicate the great importance of the endometrial spiral arterioles in the bleeding of menstruation. Their possible role in abnormal functional bleeding cannot be overlooked, nor can that of the vasomotor nerves and the uterine musculature, though much less is known concerning these factors than there is about the endocrines concerned in endometrial bleeding.

Clinical characteristics. Not much need be said as to the clinical aspects of functional bleeding. It may occur at any age during reproductive life, and occasionally outside the reproductive era. More than one-half of all cases, however, are encountered in middle life, during the so-called menopausal years, using the term "menopausal" in a broad sense as embracing roughly the fifth decade of life. It is, nevertheless, very often seen at the other extreme of menstrual life, in girls at or near the age of puberty or in early adolescence. This group, in the author's experience, constitutes about 10 per cent of all cases. The remaining 30 or 35 per cent is distributed rather uniformly throughout the intervening years of menstrual life.

The bleeding may be either menorrhagic, metrorrhagic, or a combination of the two, though the periodic type is perhaps more characteristic. In the milder cases there may be only a moderate excess of menstruation, while in others there may be such profuse and prolonged flooding as to exsanguinate the patient. In some cases the menstrual rhythm is fairly well preserved; in others the tempo is quickened; and, in still others, it is retarded, so that nonbleeding phases of considerable duration may be interposed between the floodings. This is more frequently observed in the menopausal and puberal types of functional bleeding. Again, there may be intermenstrual bleeding of varying degree, or the bleeding may be continuous and profuse for many weeks or even months.

Mechanism. The starting point in efforts to explain the mechanism of functional bleeding was the work of Schröder in 1915. By correlated histologic studies of the uterus and ovaries in such cases he concluded that the bleeding disorder to which he gave the name of *metropathia hemorrhagica* is produced by abnormal persistence of unruptured follicles, with consequent absence of functioning corpora lutea, and with the production of hyperplasia of the endometrium as a result of the abnormally persistent and excessive estrogenic stimulation. This is still the accepted explanation for perhaps the largest number of cases of functional bleeding, and for this viewpoint there is much evidence, such as the experimental production of hyperplasia in castrated animals and also in castrated human females.

Since then, however, it has been learned that not all cases of functional bleeding are to be fitted into one mold. In many, for example, the endometrium is not hyperplastic, and it may actually be atrophic. Moreover, in a considerable proportion of cases the endometrium may actually be of the progestational rather than the proliferative type, thus indicating that the cycle is of ovulatory and not anovulatory character. Little is known about the mechanism of the bleeding in cases of the ovulatory variety. A possible explanation is a quantitative imbalance between the two ovarian hormones, but perhaps dysfunction of the uterine musculature, disturbances of the vasomotor nerves, or disorders of the spiral arteriolar system may prove to be more important. It seems useless to speculate further about this group in the present state of knowledge.

The fact remains that a large, and in the author's opinion the largest, group is to be explained by the failure of ovulation, with abnormally persistent estrogen output and with the absence of corpora lutea and progesterone, in line with Schröder's original theory. No one can afford to be arbitrary in this explanation but it is, as already mentioned, supported by considerable evidence. Such a mechanism represents simply an exaggeration of that seen in the anovulatory type of menstrual cycle, the not infrequent occurrence of which is now generally accepted.

Even granting the correctness of the above explanation as to the underlying ovarian dysfunction, there remains the still unsolved question as to how this brings about endometrial bleeding. It seems probable that the bleeding phases are brought about by temporary drops in the estrogen level because of the inhibition of pituitary gonadotropic function, which is produced by more than a certain level of estrogen quantitatively different in different women. This is a very general statement and it blankets certain other imponderables, such as the receptivity or refractoriness of individual endometria to the ovarian hormones, of certain ovaries to the pituitary gonadotropes, and of specific hypophyses to the ovarian hormones.

It is now known that there is frequently a disparity between the growth and bleeding effects of the estrogenic hormone upon the endometrium. Functional bleeding can occur not only from an endometrium in which hyperplasia has been brought about by excessive estrogenic stimulation, but also from endometria in which little or no growth effect is evident, endometria which correspond perhaps to an early interval phase or which may even be atrophic. One of two explanations would seem to apply to such cases. It is probable that certain endometria are refractory to the growth effects of estrogen, although the interplay between ovarian and pituitary sex hormones still goes on, with periodic drops in the relative estrogenic levels and, therefore, with attacks of uterine bleeding. Or the bleeding spill takes place at such low estrogen levels that a growth effect in the endometrium is not produced. Hormone studies of the urine have not yet clarified this problem.

TREATMENT

The treatment of this disorder is influenced by two chief factors, the age of the patient and the importance or unimportance of preserving the reproductive function.

First, it should be remembered that no sharp dividing line can be drawn between the quantitatively normal menstrual periods generally associated with the anovulatory mechanism and the pathologically excessive bleeding in which the same underlying mechanism is concerned. Just as the girl at puberty often exhibits anovulatory cycles which by spontaneous readjustment of the hormone mechanism become ovulatory in character, so may similar spontaneous readjustment of the mechanism bring about the *spontaneous cure* of functional bleeding. This is particularly true of the milder forms of bleeding so often seen in the puberal and premenopausal periods of life.

A considerable proportion of girls exhibit not only marked irregularity but also moderate excess of menstruation for periods of from several months to several years after initiation of the function. Many of these, without treatment, later fall into a normal menstrual swing. A similar observation may be made in women approaching the menopause, a certain proportion of whom unquestionably cease ovulating a considerable time before they stop menstruating. Such anovulatory cycles may be moderately excessive. While women are properly taught that flooding at this time is never to be looked upon as normal, the fact remains that many women who disregard this teaching cease bleeding spontaneously, through the final cessation of ovarian function.

Treatment in the menopausal group. When, however, the abnormal bleeding becomes free or alarming, the patient is quite sure to seek advice. If she is in the menopausal group, the treatment is simple and highly satisfactory. At this age the first essential is to make sure that the bleeding is really functional and that no structural lesion, such as a uterine or ovarian neoplasm, is present. In the event pelvic examination reveals no evidence of this, the next step is to eliminate intra-uterine pathology and especially adenocarcinoma of the uterus. This can be done only by *diagnostic curettage and microscopic examination.* If these procedures reveal an endometrium of the proliferative or hyperplastic type, or even one corresponding to any of the normal menstrual phases, it is fair to conclude that the bleeding is of functional origin.

Once this has been settled, the further management is clear. Women at this phase are rarely anxious for further pregnancies, and practically 100 per cent of such cases can be cured by abolishing ovarian function by *radiotherapy*. This is not the place to discuss the technic of the latter procedure, but an effort should be made to give sufficient dosage to abolish ovarian function completely. Some ovaries are notoriously hard to destroy, and after insufficient dosage bleeding may reassert itself in from a few months to several years. This may be disconcerting, because while such bleeding is likely to be again of functional nature, it is difficult, without repetition of diagnostic curettage, to eliminate an intercurrent intra-uterine carcinoma. All in all, however, the treatment of functional menopausal bleeding represents one of the most satisfactory applications of radiotherapy in the field of gynecology. There is rarely any justification for hysterectomy unless there is some other indication for opening the abdomen, in which case there can be no objection to simple removal of the uterus.

Treatment in younger patients. The problem is far more difficult and unsatisfactory in younger patients, in whom the matter of future reproductiveness is usually of the greatest consequence, not to speak of the importance of preserving ovarian endocrine function. In the case of young patients curettage is not usually necessary for diagnostic purposes, and if simple pelvic examination reveals the essential normality of the pelvic organs there is little risk in assuming that the bleeding is of functional nature. Occasionally curettage is necessary for its immediate therapeutic effect, for it rarely fails to check the bleeding temporarily. It need hardly be added that the operation should be preceded by transfusion if the bleeding has been at all alarming.

It is in this group of younger patients, in whom more radical and more certain measures are contraindicated, that there is a field for such organotherapy as is now available. Radiologists agree with gynecologists that radiotherapy should, as far as possible, be avoided in patients of this group, even in carefully controlled doses designed to check the menstrual function only temporarily. In the occasional intractable case such regulated radiotherapy may be justified, though most gynecologists would probably prefer occasional repetition of curettage. Some ovaries are singularly sensitive to the effects of radium and roentgen rays, and the author has personally observed a number of instances in which even a small dosage in the hands of expert radiologists has been followed by permanent abolition of the ovarian function. Moreover, there is still no unanimity as to the effect of such treatment upon future pregnancies, particularly with reference to the possibility of defective offspring.

Organotherapy. From what has been said as to the incompleteness of the knowledge of pathologic physiology of uterine bleeding, it is not surprising that the organotherapy is still far from satisfactory. It is based chiefly on the concept that the bleeding is caused by the relative excess of estrogen and the lack of progesterone, and it aims chiefly to increase the progesterone or to lessen and counteract the estrogen. The following organotherapeutic plans may be discussed in summarizing fashion:

1. THE USE OF THE CHORIONIC OR ANTERIOR PITUITARY-LIKE HORMONES OF PREGNANCY URINE. The original employment of these substances was based on the hope that the pregnancy urine hormone principles might produce luteinization in the human ovary as they do in the ovaries of experimental animals, thus supplying the lacking hormones. It should be remembered that when this clinical experiment was made progesterone was not available for clinical use. Histologic studies of the ovaries of treated women soon established that the desired luteinization was not produced, but the fact remains that many clinicians have reported good results in a certain proportion of cases.

On the other hand, no one can claim consistent or highly satisfactory results, nor has anyone been able to explain how such substances exert their effect. It has seemed to the author that a larger proportion of cases respond to these pregnancy urine preparations than to progesterone, especially when the latter is used in small dosage. When these preparations are prescribed the usual plan is the hypodermic injection of doses of 200 to 500 units daily with the onset of bleeding, continued until bleeding is controlled or until six or eight injections are given. If free bleeding persists beyond this, not much is to be expected from further injections and other plans of treatment should be adopted.

2. THE USE OF PROGESTERONE. This hormone is readily available commercially, though still quite expensive in the dosage often indicated in the treatment of functional bleeding. Certainly no one would expect to bring about secretory changes in a proliferative type of endometrium with doses of from one to a few milligrams daily for a few days. The many reports of good results with such dosage are explained better by the assumption that the disordered bleeding mechanism is influenced by much lower dosage than is required to bring about histologic changes in the endometrium. This is in accord with what has been said in a previous paragraph as to the different thresholds of hormone sensitivity of the various endometrial functions.

3. THE USE OF TESTOSTERONE PROPIONATE. The effect of the androgenic hormones upon the female cycle has been studied extensively during the past two or three years, but there is still much that is not clear. Almost all investigators believe that the effect upon the growing follicle is an inhibitory one, and that thereby estrogen production and endometrial

proliferation are lessened. There is evidence, too, that in the progestational phase of the ovulatory cycle the effect of progesterone is supplemented and enhanced by the androgenic principle.

It is not surprising that the male sex hormone substance, in the form of testosterone propionate, recently has become popular in the treatment of functional bleeding. At the outset it has yielded more satisfactory results in the author's experience than either progesterone or the pregnancy urine preparations. During the bleeding phases it may be given intramuscularly in doses of 10 mg. three times a week, and in the more severe cases 25 mg. twice a week. Much larger doses are used by some, but the author believes that this tendency should be avoided. A number of reports have been published of hirsutism and occasionally other masculinization phenomena, such as a change in voice. These symptoms apparently disappear upon withdrawal of the drug, though very slowly.

In any event, such sequelae are highly undesirable, and if they were at all common the use of testosterone would scarcely be justified. The author does not believe the moderate dosage suggested herein is attended with any worthwhile risk of these unpleasant symptoms, especially if it is avoided in patients who already show a tendency to hirsutism or excessive pigmentation. The best policy is to avoid the larger doses and rarely to give more than 50 mg. a week, especially when, as in cases of functional menorrhagia, the hormone is kept up throughout the cycle. In these early days of testosterone therapy it is advisable to be constantly on the alert for undesirable by-effects, such as those mentioned, until further experience enables a more intelligent evaluation of the safety or the hazard involved.

4. The use of thyroid extract. In only the occasional case of functional bleeding does hypothryoidism appear to be a factor but, when it is, thyroid therapy is indicated and is likely to be curative. A striking case of this type was recently observed. The patient was a woman of thirty-two who, for several years before the author saw her, had been taking exceptionally large doses, 20 grains a day, of thyroid extract, with no evidence of saturation symptoms, and with a basal metabolic rate still slightly on the minus side. If this dosage was stopped or markedly lessened profuse menorrhagia would always appear, with the return to a normal menstrual amount upon resumption of the thyroid extract. In the overwhelming majority of cases of functional bleeding, however, no thyroid element appears to be concerned and thyroid therapy is of no value.

5. Other measures. Hamblen has recently reported good results in functional bleeding from what he speaks of as the cyclic employment of estrogens and progesterone, or of progesterone alone. This method seems too new to permit of evaluation. Pregnant mare serum preparations have

likewise been tried, but according to the yet meagre reports available have not yielded noteworthy results. It need hardly be stressed that general *building up measures* are never to be overlooked, and that *hematinic treatment* and not infrequently *transfusion* are often required. The author has, in several papers on the general subject, mentioned the desirability in the occasional cases in which this is practicable of using for transfusion the blood of pregnant women.

NEAR-ULTRAVIOLET RAYS AND FLUORESCENCE PHENOMENA AS AIDS TO DISCOVERY AND DIAGNOSIS IN MEDICINE* † ‡

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The development within recent years of practical, efficient sources of near-ultraviolet radiation—the invisible energy which causes many materials to fluoresce or emit visible light—has made possible another extension of the limited range of visual sensitivity. Simple effective methods and apparatus for utilizing near-ultraviolet radiation and fluorescence phenomena to disclose facts that are not discernible in ordinary visible light will be described and discussed in this paper. The purpose of this will be to stimulate the more general use of fluorescence phenomena in all types of medical examinations, from routine studies of patients to postmortem and microscopic examinations. The procedures and principles involved are not new and, while they have been employed extensively in other fields to discover many strange and interesting facts, their application in the field of medical examination has been recent and limited. This is, therefore, almost a virgin field and many discoveries and spectacular observations await those who can intelligently and consistently use fluorescence to aid and supplement their powers of perception.

The physical concepts and definitions will be discussed briefly. This will be followed by a discussion of the necessary apparatus and conditions, including evaluation of possible dangers and precautions that should be taken. The more common applications and discoveries in other fields will be mentioned, while the known applications in the fields of medical diagnosis and physiologic chemistry will be treated more fully. Lastly, the author's observations on normal individuals will be recorded in the hope that these will be suggestive and of value for comparison with records obtained by routine examination of patients.

DEFINITIONS AND PHYSICAL CONSIDERATIONS

Many substances have the property of absorbing radiant energy, usually over a characteristic range of wave lengths. This is converted into chemical energy or radiant energy of the same or longer wave length, such as heat or visible light. If a piece of filter paper is moistened with a solution of quinine sulfate and illuminated with the invisible ultraviolet portion of the solar

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† From the Department of Anatomy, School of Medicine, University of Maryland.

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spectrum, it will emit a brilliant blue visible light. Stokes (31) in 1852 applied the term *fluorescence* to this phenomenon because it was first observed in fluor spar or fluorite. When the exciting incident radiation is cut off, the fluorescence ceases. If a substance continues to emit light for some time after the external exciting radiation has ceased to act, it is said to be phosphorescent. Photoluminescence, which includes both fluorescence and phosphorescence phenomena, is the absorption and reradiation of radiant energy by a substance. It is highly probable that the only difference between fluorescence and phosphorescence is in the duration of the after-glow.

Detailed accounts concerning the theoretic aspects of the nature of light and the theories of light absorption and emission are beyond the scope of this work and may be found in the recent books on fluorescence analysis (5, 28). At the present time the most generally accepted theory of fluorescence is related to the quantum theory and Bohr's theory of the atom. According to these theories, fluorescent substances, when illuminated with rays in the near-ultraviolet region of the spectrum, absorb these rays. Electrons are thereby displaced from their orbits, probably to orbits farther away from the nucleus. This establishes an unstable condition and stabilization involves the return of the electron to the original orbit or level. Such a process results in the emission of radiation. The wave length of this emitted radiation need not be the same as the exciting radiation, but depends rather on the electron displacements involved. As a matter of fact, it is a general rule that the exciting radiations are of shorter wave length than the emission radiations. This general rule, which is known as Stokes' rule, was discovered long before the existence of the quantum theory. It substantiates this fact because the rule indicates that one cannot expect more to come out of the atomic mill than goes into it.

These general theories are probably applicable to all parts of the electromagnetic spectrum, but for the purpose of this discussion need only be applied to the near-ultraviolet and visible regions. Although not entirely satisfactory, they explain how substances that are visibly fluorescent absorb the invisible near-ultraviolet radiations and emit visible light of longer wave length. It is perhaps a fortunate circumstance that a complete and satisfactory understanding of these phenomena is not a prerequisite for their application and utilization.

After the discovery of fluorescence by Stokes (31) the studies utilizing this phenomenon were for some time sporadic and characterized by isolated, nonrelated observations in many fields. For a long period fluorescence was regarded mostly as a scientific curiosity and used chiefly for spectacular demonstrations in physics laboratories. Then followed a period during which fluorescence was used by theatrical and advertising agencies in the form of impressive stunts to amaze the layman. Most of this work had no value from the scientific viewpoint, but it served to stimulate the development of practical sources of near-ultraviolet radiation.

The discovery and development of methods to utilize fluorescence phenomena for ordinary illumination, the so-called *fluorescent lighting*, coincidently accelerated the perfection of near-ultraviolet sources. The fluorescent lights are becoming increasingly popular because they are far cooler, much more efficient, and a more uniform light source. They are about four times as efficient as the tungsten filament light. These tubular lamps generate ultraviolet radiation which is absorbed and retained inside the tube by an inner coating of a substance that is highly fluorescent. This substance then emits radiations in the visible range. By using a tube of near-ultraviolet filter glass containing nickel and cobalt, but without an inner coating, such lamps are converted into lamps that may be used for fluorescence studies. Because of the commercial developments these lamps are now available for scientific work at relatively low cost. Some confusion may arise from the fact that the lamps designed for visible illumination by fluorescence are called fluorescent lights. It is, therefore, important to remember that the fluorescent lights coming into popular use radiate visible light, while the so-called black lights or lamps used for fluorescence studies radiate invisible near-ultraviolet light.

APPARATUS AND OTHER REQUISITES

For the purpose of exciting and observing fluorescence the most effective radiation is invisible or near-ultraviolet light. This includes only the wave lengths between 3100 and 4000 Angstrom units. There are some lamps, such as the argon glow lamp, that are excellent sources of near-ultraviolet. They are inexpensive and do not require accessory filters since they produce only a small amount of visible light. The intensity of the light produced by these argon glow lamps is so low that they are only suitable for the examination of a limited number of highly fluorescent materials. They are not suitable for routine examinations of materials and objects of variable and unknown fluorescence intensity.

In order to secure a near-ultraviolet light source intense enough to be practical for fluorescence studies it has been found necessary to use quartz mercury arcs or carbon arcs that exhibit high radiation characteristics in the visible and both far- and near-ultraviolet portions of the spectrum. The visible (4000 to 7000 Å) and far-ultraviolet (2000 to 3100 Å) wave lengths are undesirable and relatively ineffective for exciting fluorescence. These wave lengths are, therefore, eliminated by the use of filters that selectively absorb both far-ultraviolet and visible light and transmit an intense band of near-ultraviolet light.

Many lamps which utilize glass filters to isolate a band of near-ultraviolet are available at present. Some of these, for various reasons, are not suitable for use in medical and biologic work. The intensity of the near-ultraviolet radiated by most of the older and less expensive lamps, as in the case of the argon glow lamp, is not great enough to excite much fluorescence. Old type lamps with sufficient intensity are usually expensive, as well as clumsy and inconvenient. In other lamps the filters used to isolate the band of near-ultraviolet light do not really isolate this band, but transmit appreciable amounts of visible light. This may completely mask or be confused with fluorescent effects if such a filter transmits red light in addition to near-ultraviolet, as many of them do. For example, blood which ordinarily does not fluoresce appears almost black in near-ultraviolet. In the light from a filter that transmits both near-ultraviolet and red light, blood appears bright red. When objects or materials that exhibit red fluorescence are examined in light transmitted by such a filter it is impossible to distinguish between the red light caused by the fluorescence and that resulting from reflection.

The ordinary therapeutic quartz mercury arc or carbon arc lamps in general use by dermatologists could be converted into lamps to study fluorescence. This would merely require the purchase of proper glass filters* to absorb the visible and far-ultraviolet and transmit only the near-ultraviolet to the object to be examined. Since some substances or objects that exhibit weak fluorescence cannot be detected in the presence of any visible light, such filters are practical only if they can be attached in a manner that will prevent stray beams of visible light. This is extremely difficult and often impossible with some of the therapeutic lamps in use today. While the method is feasible in some instances, it is not recommended. The converted lamps are usually neither efficient nor convenient and the cost of such makeshift equipment approaches that of a simple, portable, efficient lamp available commercially and designed specifically for fluorescence studies.

For medical and biologic fluorescence studies the lamp which is recommended consists of a small 100 watt type H quartz mercury arc, surrounded by a mantle of filter glass that absorbs the far-ultraviolet and visible light and transmits only the near-ultraviolet.† It would not be necessary to eliminate the far-ultraviolet portion of the quartz mercury arc spectrum (2300 to 3100 Å) for fluorescence studies; but from the safety aspect this is a decided advantage as these wave lengths include the tanning rays, the bactericidal rays, and rays that induce conjunctivitis. Since the lamp which is recommended eliminates these deleterious far-ultraviolet rays by absorption, there is little or no danger of overexposure or bad effects either to the operator or the patient. Definite cases of sensitivity to this portion of the spectrum cannot be cited and may not exist. The author has used such a lamp sometimes for periods as long as six to eight hours a day without wearing goggles or other protective devices. Two years have elapsed and no ill effects have been observed. In spite of this theoretical and observed margin of safety, patients should be protected with appropriate goggles or at least instructed to close the eyes during examinations, except during the short intervals necessary for the examination of the eyes. In addition to this precaution, a constant vigilance should be maintained to detect as early as possible the occasional individual who might be sensitive to near-ultraviolet light.

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* Corning G 586 AW or 587.

† These lamps are available commercially from the General Electric Vapor Lamp Co. and from Westinghouse, type BH4. The outfits consist of a transformer and reflector, and are usually designated as "black light outfits." They utilize a quartz mercury arc bulb surrounded by a mantle of near-ultraviolet filter glass. The entire assembly costs approximately $30. The wave lengths of radiations from the lamp are between 3100 and 4000 Å. Most of the radiations from the light are about 3650 Å. (A negligible amount of visible violet light is transmitted.) This bulb is mounted in a reflector that may be moved easily or held or aimed in any direction. The transformer that operates this mercury vapor arc may be plugged into any ordinary alternating current outlet.

In addition to the inexpensive lamp radiating almost pure near-ultraviolet light, a dark or semidark room is the only other requirement for fluorescence studies that involve the examination of large objects. If a dark room is not available, the installation of dark curtains to exclude most of the visible light is usually satisfactory.

Fluorescence phenomena may be photographed with slight modifications of ordinary photographic technic. It is necessary to use long exposures and to protect the film from reflected near-ultraviolet light. This may be done by placing a Corning Noviol O No. 306 filter in front of or behind the lens of the camera. This prevents fogging of the film because the filter transmits only the visible light and absorbs stray ultraviolet light. The picture which is thus obtained is one of true fluorescence and designated by some authors as a "luminogram." These modifications and technics are discussed at length in books on fluorescence analysis (5, 28, 29) and works on photography and ultraviolet light (4).

APPLICATIONS

Only a few of the industrial and scientific applications utilizing invisible or near-ultraviolet radiation will be mentioned. For more extensive accounts the reader is referred to more comprehensive manuals. The two books from which most of the references are chosen are both on fluorescence or luminescence analysis. One of these (5) appeared in 1928. The other is in English (28) and appeared in 1933. Both of these books cover the same material and in approximately the same way. As indicated by their titles, most of the work has been confined to the detection, identification, and quantitative determination of drugs and chemicals. However, many of the applications are in other fields.

Museums, art galleries, and art collectors have used near-ultraviolet to determine the age and authenticity of many art treasures. In the case of a picture attributed to Pissaro, there appeared the fluorescent outline of another signature which had been removed before that of Pissaro was substituted (21). In another case, the signature on a Ruben's picture was questioned because it was indistinct in visible light. The signature showed very clearly in ultraviolet light. In addition to forged and authentic signatures, repairs and overpaintings can sometimes be detected easily (2). Some pigments show fluorescent colors that vary considerably, but varnishes are so fluorescent that they may obscure the fluorescence of the pigments under them (4). Freshly cut marble fluoresces an intense purple which is uniform. As it ages it develops white, yellow, and blue tones and a mottled appearance. The method has been used to indicate the age of marble, alabaster, and ivory carvings and statues (29). Newly cut ivories may be made to appear old in visible light by bringing them in contact with manure, skin, or tobacco juice, or by allowing a turkey to swallow them, but these artificially aged objects are discovered readily in near-ultraviolet light.

Laundries are utilizing near-ultraviolet light and fluorescent inks to facilitate the rapid sorting of garments and to avoid marking fine linens and other articles with ordinary visible ink. Since different inks, glues, and papers fluoresce in characteristic ways, this light is used by banks, detectives, and criminologists to discover many things. Forgery, the alteration of documents, and mail that has been tampered with are often detected by simple examination. It is also of use in criminologic work to detect and help identify blood stains on garments (35). Many of the methods used in this work convert hemin to porphyrin, which has a characteristic red fluorescence. In rape cases, spots caused by seminal fluid on garments or other materials are located easily because of strong blue-white fluorescence (20). These are then examined microscopically. The detection of spermatozoa serves to confirm a positive fluorescence indication. Stains caused by urine have a more yellow fluorescence than those attributable to semen.

In the field of public health and agriculture these rays have been used to determine the freshness of food (eggs, meat, walnuts); to distinguish between butter and margarine (30); to detect horse fat in lard and chickory in coffee. Many fungus infections and other plant diseases may be detected by fluorescence. The spread of ring rot in potatoes is being prevented by cutting seed potatoes under near-ultraviolet light. When a green fluorescent potato is encountered it is discarded and the knife disinfected.

Hair containing Microsporon or Tricophyton also fluoresces with a bright green color. This fact is now being used for the detection of ringworm infection (14, 18, 22). Psoriasis scales and hyperkeratose scales both fluoresce white, but in the case of psoriasis they have a more chalky fluorescence (5, 28). Stubel (34) was one of the first to study animal tissues. He showed that all animal cells and tissues have the ability to fluoresce in ultraviolet light. The presence of nonfluorescent pigment or hemoglobin usually minimizes this. Bommer (3) has described the appearance of the fluorescence of inner organs. He mentions the white fluorescence of bones and that cartilage is bluish-white. Connective tissue (fascia) exhibits an intense blue-white, so also do the dura and pia. The skeletal musculature is described as fluorescing dark brown, fat as light yellow, and the heart muscle as yellow-brown. The wall of the uterus in section appears yellowish-gray. The fluorescence of the glandular organs is described as follows: thyroid, reddish-gray; carotid body, definite red; adrenal, orange-red; pancreas, brownish-yellow; testis, dark gray-red; liver, an intense yellowish-green to brownish-yellow. The lens, sclera, and the cornea all fluoresce; however, the pale but strong bluish-white fluorescence of the lens is most conspicuous.

The fluorescence spectra of a number of the carcinogenic agents were found to be similar and confined to the band between 4000 and 4400 Å. Kennaway and Hieger suggest this as a method of detecting carcinogenic substances in commercial coal tar and oil products (17). They point out that mice must always be used to prove conclusively that the substances so detected are carcinogenic. Fluorescence phenomena might be useful for the early diagnosis of cancer. This suggestion was made on the basis of the fact that the addition of sera from normal men or horses does not change the fluorescence intensity of uranin, but the addition of sera from cancer patients reduces it in intensity (1).

Most of the work on the fluorescence of blood and blood derivatives concerns itself with porphyrins. These substances exhibit such a strong and characteristic red fluorescence that the literature on this is extensive. This red fluorescence was mainly responsible for the detection of porphyrins in the harderian glands of rats, abscesses, amniotic fluid of sheep, the quills of young porcupines, and the teeth of some young mammals (6 to 11). Policard (24, 25, 26) has found that certain malignant tumors, sarcomas, exhibit a red fluorescence that is related to the presence of hematoporphyrin. The human ovary also was examined with a fluorescence microscope by Policard (27). The stroma appeared white with a bluish tinge. The corpora lutea stood out with a yellow fluorescence. The region of the follicles did not fluoresce, but was black and opaque. The atretic follicles were surrounded by a yellow fluorescent line and the follicular cavity was filled with a vitreous blue transparent mass.

Many more examples of discoveries that were made by using near-ultraviolet light to detect fluorescence could be given. Those which have been discussed will suffice, however, to indicate the rapid advances that might result from the general use of near-ultraviolet for routine examinations. Many of the references were selected because of the direct or indirect relationship that they have to the following fluorescence studies by the author.

During experiments* on the reduction of estrone† to estradiol these two substances were dissolved in a 1 molar sodium hydroxide solution containing about 1 per cent ethyl alcohol. Such a solvent exhibited no fluorescence. The estrone solution exhibited a brilliant light green fluorescence, whereas the estradiol solution fluoresced a deep blue. By means of filters it was possible to see that the estradiol solution was also green fluorescent, but that this was masked by the intense blue. Likewise, the estrone solution showed some blue fluorescence which was masked by the brilliant green. The estradiol solution gradually lost its deep blue fluorescence on standing and became blue-green. The green fluorescence of estrone in concentrated sulfuric acid had been described previously (19). No reference to this differential fluorescence of estrone and estradiol in 1 molar sodium hydroxide was found in the literature.

One of the most striking and spectacular examples of fluorescence of animal tissues was the brilliant scarlet red fluorescence of the harderian gland of the rat. When the eyeball was removed from a rat, a gland which appeared fatty and slightly brown in visible light filled the rest of the orbital cavity. Under near-ultraviolet light this whole gland emitted a brilliant red glow. A rat without eyes in this light appeared to have balls of red fire in the orbital cavities. This observation was made during an attempt to dissect a nasolacrimal duct under near-ultraviolet light. At the time, an attempt was being made to discover the glandular source of the red fluorescent exudate which appeared on pantothenic acid-deficient rats (23). Later it was shown by an ablation experiment that this gland was the source of the red fluorescent porphyrin-containing exudate that appeared on the noses of pantothenic acid-deficient rats (12). While this rat harderian gland fluorescence was noted independently, it had been observed and described long ago by Derrien and Turchini (6). In addition, these workers had related the red fluorescence to the presence of protoporphyrin. The fluorescent harderian glands observed with a fluorescence microscope had also been described by Grafflin (15).

The spectacular fluorescence of the harderian gland was so impressive that it inspired the examination of a number of other animals. Other rodents have harderian glands but, strangely enough, these do not fluoresce red. No red fluorescence was observed in the contents of the orbital cavities of monkeys, dogs, cats, rabbits, guinea pigs, squirrels, or an opossum. Mice of two inbred strains were available from another experiment on cancer susceptibility (13). It was observed that the harderian gland of a mouse of the mammary cancer-susceptible strain was red fluorescent, but this same gland from a mouse of the mammary cancer-resistant strain was not red fluorescent. This observation was extended to a large number of mice in sixteen different strains (32, 33). In general, the original observations were substantiated with certain minor modifications resulting from age variation. There was a close parallelism between the degree of red fluorescence of the harderian gland and the degree of susceptibility to spontaneous mammary carcinoma.

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* These experiments were started at Yale University Medical School during a year's leave of absence as a Rockefeller Fellow, 1940–41.

† The estrone was kindly supplied by Parke, Davis & Co.

During the early part of this work on red fluorescent porphyrin in animal tissues and exudates a circus advertisement seen many years ago was recalled. In this the hippopotamus was described as a "blood-sweating behemoth." Other books on natural history mention this bloodlike secretion of the hippopotamus, though they point out that it is not really blood. It was thought possible that this red or pink secretion or exudate might be a porphyrin secretion of the sebaceous glands. The hippopotamus* was, therefore, illuminated at night with near-ultraviolet light. No red fluorescence was observed. A small amount of the red material was collected for laboratory examination. The fact that it was red was substantiated, but it did not fluoresce. This indicated that the red material was either not a porphyrin or that it existed in some complex form that was not fluorescent.

It was noticed during the casual examination of colleagues that the so-called blackheads or comedones were red fluorescent. It was found that these secretions of the sebaceous glands could be squeezed out and placed on slides, where they exhibited a red fluorescence similar to that observed in the harderian gland. This gave rise to the hypothesis that perhaps the harderian gland and the sebaceous glands might be closely related. Some evidence has been found to support this view. It was, therefore, desirable to make similar examinations on a large number of normal individuals to see if there would be any variation in the red fluorescence of sebaceous secretions comparable to the variations observed in fluorescence of the harderian glands of mice.

This survey has been started by examining medical students and, while it is far from complete, great variations in the red fluorescence of comedones have already been observed. Out of the fifty individuals examined only two had no red fluorescent comedones. On the other hand, some individuals had so many red fluorescent comedones that almost the entire nose and face took on a red glow in near-ultraviolet light. The observed variation is difficult to describe in terms of the intensity of the red fluorescence; however, this appeared to be related in a definite way to the number and distribution of red fluorescent comedones. In the cases showing a maximum intensity of red fluorescence there were thousands of small red fluorescent spots on the face and forehead. The largest and reddest spots were found at the base of the nose and on the nose. Large numbers of red fluorescent spots also were seen on the forehead, chin, and cheeks in these instances. A gradient with regard to both the intensity of fluorescence and the number per unit area usually was observed. In the cases of maximum fluorescence, the red spots, in addition to those mentioned on the face and forehead, may be quite numerous on the neck, shoulders, and pectoral regions and extend as far caudalward as the lumbar and abdominal regions. The number and extent of the distribution may be used as a substitute for a method to determine the intensity of the red fluorescence.

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* The author is indebted to the officials of the Druid Hill Park Zoo, Baltimore, for their courtesies and splendid cooperation.

In most of the individuals examined there was a variable number of red fluorescent spots near the base of the nose, on the chin, near the angles of the mouth, in the ears, and on the forehead. They were less numerous on the cheeks, and white or yellow-white fluorescent comedones were interspersed among the red ones on the cheeks and neck. In passing to more caudal regions the white fluorescent comedones increased in number per unit area and the red ones decreased.

So far no reference to this red fluorescence of comedones has been found in the literature. The red fluorescence is probably caused by the presence of a porphyrin, but this, as well as the significance of the observation, must be left for future study.

In connection with the survey of red fluorescence phenomena in normal individuals some interesting data on the oral cavity have been obtained. In most individuals the dorsal half of the upper surface of the tongue fluoresced a deep shade of red; in others this was orange-red; and in a few it was orange or yellow and inconspicuous. A variable portion of the tip and margin of the tongue was found to exhibit a dull blue-gray fluorescence. Since these observations were made it was found that the orange fluorescence of the tongue had been described (5, 28). The variations in color were not mentioned, however. Thus far no significance can be attached to these variations in the color of the fluorescence of the dorsal half of the tongue.

Approximately one-fourth of all subjects examined had red fluorescent tartar on the teeth. The medical students submitting to the examination were requested not to brush their teeth on the morning of the examination or the previous evening. This red fluorescent tartar was most abundant on teeth that were probably fundamentally poor, as indicated by numerous fillings and erosion. Sometimes it appeared only on one or two teeth. It has been shown by Hymans van den Bergh (16) that this red fluorescence is attributable to the presence of porphyrin in the tartar. It is also said to be more common on the teeth of older individuals. Whether this porphyrin causes bad teeth or is the result of them, or whether its association with bad teeth is coincidental, are also problems for further research.

SUMMARY

The principles and requisites for the use of near-ultraviolet for fluorescence studies are reviewed and discussed. The applications in many fields are mentioned to emphasize the potentialities and versatility of this method of extending the range of visual sensitivity. The observations of the author on the fluorescence of sex hormones, harderian glands of rats and mice, human comedones, and the tartar on teeth are described briefly. The use of near-ultraviolet and fluorescence phenomena in medical and biologic investigation is still in its infancy. A more general and systematic utilization of this method can only yield unpredictable dividends in discoveries and scientific advancement.

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CONGENITAL ENCAPSULATED MULTILOCULAR SEROUS CYST OF THE KIDNEY ASSOCIATED WITH HYPERTENSION; OCCURRENCE IN A NINETEEN MONTHS OLD INFANT*†

Report of a Case

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This paper is a report of an unusual case occurring in a nineteen months old infant of a multilocular encapsulated serous renal cyst, associated with sustained hypertension and a clinical and roentgenologic picture which suggested a renal embryoma (Wilms' tumor).

In adults the frequency of small cortical cysts in the arteriosclerotic kidney is well known. Solitary cysts, multiple cysts, multilocular cysts and congenital polycystic disease are less common. Polycystic kidneys have been reported by Bell (1) to occur about once in 3523 hospital admissions or, roughly, once in every 500 autopsies. Solitary renal cysts have been studied by Harpster (2) who found but thirty-four cases in 4250 autopsies. In children, Harpster found only three instances of solitary renal cysts in individuals less than four years of age. Bugbee and Wollstein (3) found twenty cases of renal cysts in a series of 4903 autopsies performed on children, 101 of whom were under one year of age. Their studies revealed polycystic disease in fifteen instances and hydronephrosis with cysts in three instances, solitary cysts being found in only two cases. Campbell (4) has thoroughly reviewed the subject of renal cysts in children and brought the literature up to date, but no mention is made of multilocular encapsulated serous cysts. In a search of the literature covering renal cysts no reported cases of encapsulated multilocular serous cysts in children have been found.

The etiology of reported types of renal cysts remains vague. In adults, solitary serous and hemorrhagic cysts have been regularly associated with some accompanying kidney lesion and Balch (5) reports that 35 per cent are associated with either infection, a neoplasm or a degenerative process. Hepler (6) has shown experimentally that solitary serous cysts may be produced by a combination of trauma and an altered blood supply. The theory of Kampmeier relating to the development of polycystic disease of the kidneys is generally accepted and will probably survive. Other rarer congenital cysts have been recorded, such as congenital hydrocalycosis

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(12), congenital hydronephrosis with multilocular cysts, and metanephric cysts, as reported by Strauss and Krauss (7). The most uncommon cysts found in children are congenital multilocular cysts. Virchow, Laveran (9), Campbell (4), and Geisinger (10) have reported the occurrence of these cysts, but emphasize the fact that they intend these multilocular cysts in no way to be confused with congenital polycystic disease.

Bell (1), Cabot (8), and others have reported what they believe to be true unilateral polycystic kidneys, but the weight of opinion is still in favor of the bilateral nature of the disease.

CASE REPORT

C. W. (History No. 43188), a 19 months old white male, was admitted to the pediatric service of the University Hospital on July 17, 1941 because of a mass in the left side of his abdomen, which was first noticed by his mother two weeks before admission. There had been no other unusual observations, and the child had not changed in appearance since the discovery of the mass.

The family history was noncontributory. Birth of the patient was premature at seven months, the birth weight being 4½ pounds. After seven weeks of routine care for premature infants in the hospital he began to gain normally. Growth and development thereafter were satisfactory. In the summer of 1940 he suffered an attack of diarrhea and during the month of June, 1940 he received several roentgen treatments for what was apparently an enlarged thymus gland. He had an attack of chickenpox in April, 1941.

On admission the patient was seen to be a normally developed white male infant. His skin possessed a sallow and almost icteric tinge; however, the texture was normal. There were no abnormalities of the head, but a slight cervical adenopathy was noted. The chest was clear and the heart normal. An electrocardiogram showed no abnormalities. In the left upper quadrant of the abdomen, under and below the costal margin, there was a firm, nonmovable mass about 10 cm. in diameter. This mass was not tender and was not freely movable. There were no other significant abdominal findings. The reflexes were physiologic. Extremities and genitalia were normal. An examination of the fundi revealed no abnormalities. The blood pressure was 130 mm. of mercury systolic and 80 mm. of mercury diastolic.

Laboratory studies. The urinalysis was normal, except for occasional white cells. Examinations of the stools were negative for ova and parasites. There was no reaction to 0.01 mg. old tuberculin given intradermally. The serologic test for syphilis was negative. Examination of the blood revealed hypochromic anemia, the color index being 0.8. White blood cells numbered 16,400, of which 46% were polymorphonuclears and 52% lymphocytes. Smears for blood parasites were negative. The platelet count, bleeding time and clotting time were normal.

Blood chemical studies showed an icterus index of 7.5, with slight indirect van den Bergh reaction. The nonprotein nitrogen was 48 mg. %.

Roentgenologic studies. A large soft tissue mass was revealed in the left upper quadrant of the abdomen. An intravenous pyelogram (Fig. 1) showed the right kidney pelvis to be normal, the left being displaced downward and medially, and the calices superimposed one upon the other. The ureteropelvic junction was filled with dye and was sharply angulated. No other part of the left urinary tract contained dye. On July 1 cystoscopy and retrograde pyelography were done. There was no evidence of infection, but the left kidney appeared practically functionless. Roentgenograms showed the left ureter and

FIG. 1

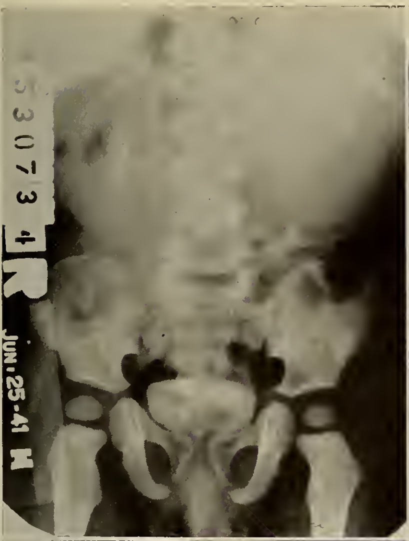

FIG. 2

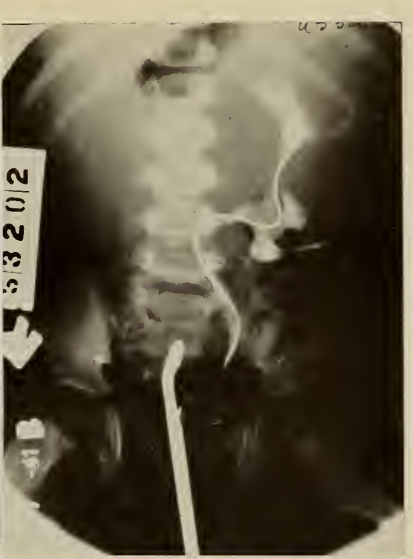

FIG. 3

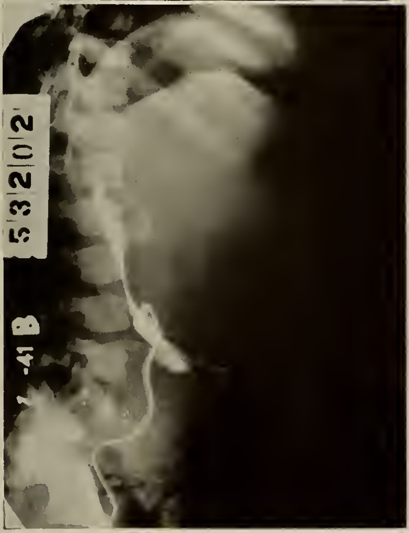

FIG. 4

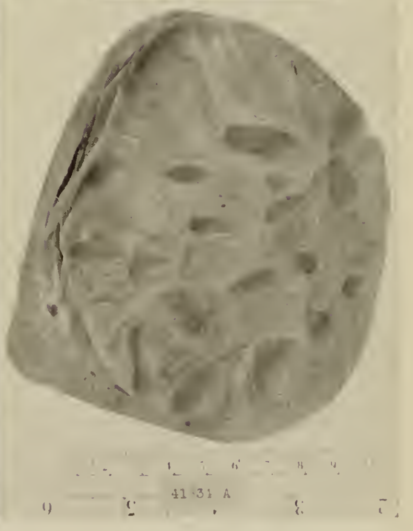

FIG. 1. Intravenous pyelogram showing a large soft tissue mass in the left upper portion of the abdomen.

FIG. 3. Retrograde pyelogram, lateral view.

FIG. 2. Retrograde pyelogram, anterior-posterior view, showing distorted calices and outline of the mass.

FIG. 4. Cross section of the mass showing an encapsulated multilocular serous cyst and a normal appearing but distorted kidney.

kidney pelvis to be well filled following a retrograde injection of radio-opaque solution. The kidney pelvis and calices were greatly distorted and displaced posteriorly, medially and inferiorly. The distorted pelvis and calices apparently delineated the posterior and inferior borders of the soft tissue mass in the left abdomen (Figs. 2 and 3). These findings were considered compatible with those of a renal embryoma (Wilms' tumor).

The child was then transferred to the urologic service and the definite impression of a renal neoplasm, possibly a renal embryoma, was confirmed.

In preparing the patient for operation, deep roentgen therapy was begun on June 30 and continued through July 16. Doses of 424 roentgen units were given through each of three portals, the left lateral abdomen, the left anterior and left posterior abdomen. It was noted that during the period of roentgen therapy the blood pressure fell gradually from 130 to 90 mm. of mercury systolic and from 90 to 60 mm. of mercury diastolic, where it remained at the time of operation. During the period of roentgen therapy the abdominal mass had not decreased in size.

On July 31, under general anesthesia and through a transperitoneal approach, a large mass was seen incorporated within the left kidney. A left nephrectomy was performed easily with no adhesions between the capsule of the mass and the surrounding fascia. There were no operative complications.

Following the operation there were moderate symptoms of shock and a rather high fever. There was slight nitrogen retention, the highest blood nonprotein nitrogen being 55 mg. %. On the third day the blood nonprotein nitrogen returned to normal. A considerable degree of abdominal distention developed, which was associated with continued vomiting and fever. By the sixth day definite signs of intestinal obstruction appeared. An emergency laparotomy was performed and a loop of small intestine was found to be kinked and gangrenous. This was resected successfully. After considerable difficulties over a period of five days, the patient rapidly improved and was discharged from the hospital on August 27, 1941. The urinalysis at this time was normal, as was the blood nonprotein nitrogen. The blood pressure at the time of discharge was 90 mm. of mercury systolic and 60 mm. of mercury diastolic. In every way the child appeared fully recovered.

Pathologic report. The specimen consisted of a left kidney, within the capsule of which was incorporated a large, firm, cystic mass measuring 12 by 10 by 8 cm. The kidney was compressed, thinned out and markedly elongated. On sectioning, this mass was seen to be an encapsulated smooth-lined multilocular cyst and measured 11.5 by 8 by 7 cm. There was compression of the kidney substance everywhere except at the hilus, where a small triangular area of recognizable and apparently normal kidney substance was found. The compression of the kidney substance became more distinct on either side of the hilus. There was marked stretching and distortion of the pelvis and calices. The ureter was patent. Pressure on the cyst did not force fluid into the pelvis (Fig. 4).

Microscopic sections through the cyst walls showed a multilocular structure, each locule being lined with a layer of compressed cuboidal epithelium. Some blood vessels were seen within the fibrous supporting stroma, and a few red cells and amorphous material were found within the cysts. Sections through the capsule of the cyst along the lateral border of the mass showed a thick fibrous capsule and adjacent to it a narrow zone of compressed renal substance. In this zone were seen remnants of compressed tubules and glomeruli (Figs. 5 and 6). There was much fibrosis and little evidence of vascularity.

A section through the grossly normal renal tissue near the hilus showed a typical picture of an infantile kidney, which was histologically normal. In the midzone, that is, the portion of the kidney lying between the hilus and the compressed and fibrous portion adjacent to the lateral cyst wall, sections showed considerable compression of the renal substance but little evidence of fibrosis. There appeared to be an adequate blood supply. Large

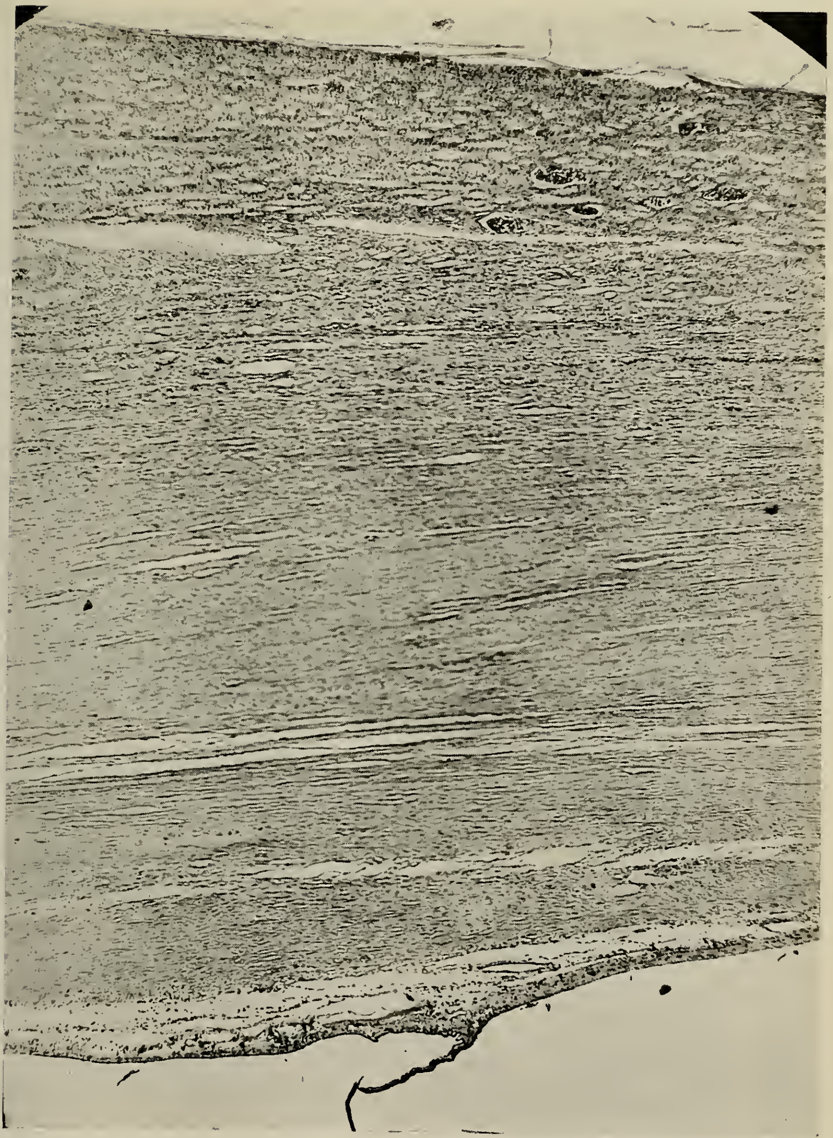

FIG. 5. Low power magnification showing capsule of the cyst and trabecula with the compressed zone of renal tissue lying without.

Fig. 6

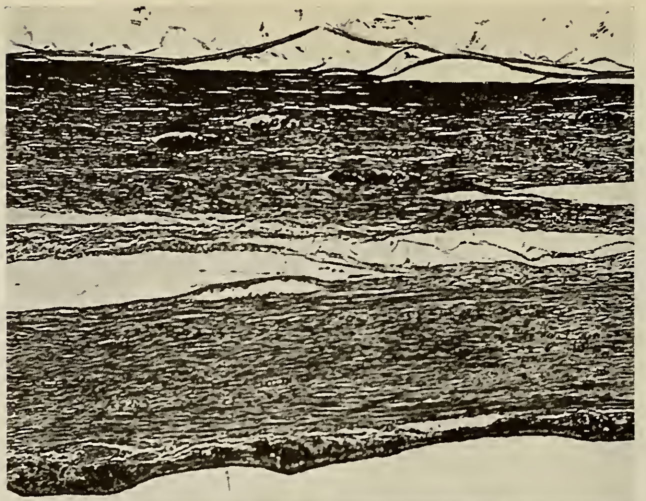

Fig. 7

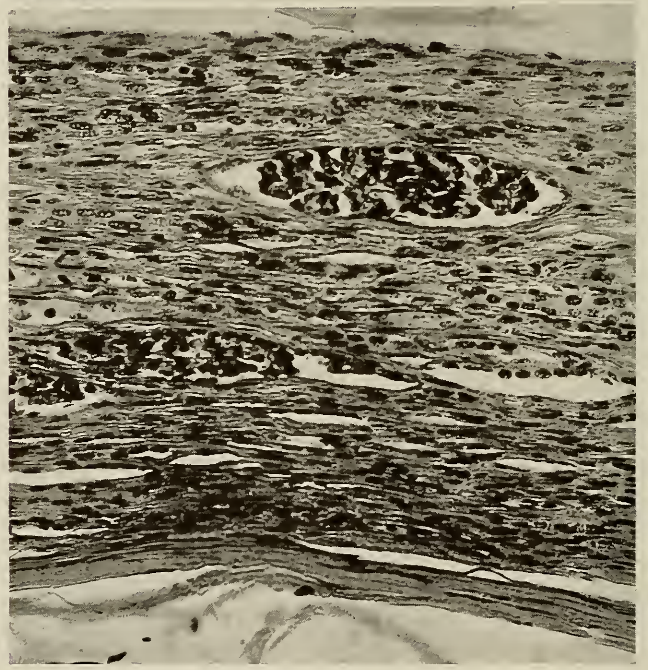

Fig. 6. High power magnification of the compressed zone in the lateral portion of the mass showing extreme fibrosis, with absence of the blood vessels and rudimentary glomeruli and tubules.

Fig. 7. High power view of the compressed but apparently functional renal tissue. The glomeruli contain blood and the capillaries appear normal. The tubules are compressed.

vessels were compressed but not thickened. The true kidney capsule was normal. There were no abnormalities of the calices (Fig. 7). This portion of the kidney, while compressed, appeared functional.

A chemical analysis of the cystic fluid showed total proteins of 223 mg. %, chlorides (as Cl) 150 mg. %, and nonprotein nitrogen 32 mg. %.

Dr. Carl L. Davis, Professor of Anatomy at the School of Medicine, University of Maryland, was kind enough to review the specimen. His report was as follows.

"One may assume that the kidney was of a normal type containing a multilocular encapsulated serous cyst incorporated within the kidney capsule, arising presumably from the cortical or the outer medullary portion. It was hardly possible for a mesonephric rest to be incorporated within the metanephros either as a remnant of the wolffian body or as a remnant of the mesonephric blastema.

"Therefore, there was but one explanation, that is, that the cyst arose from within the developing metanephros, possibly from one or more of the generations of developing uriniferous tubules, with failure of fusion and resulting cyst formation. This mechanism would therefore be in agreement with the theory of Kampmeier, except that the process was not diffuse throughout the entire developing metanephros but limited to a relatively small nest of tissue, which later became incorporated within the body of the normally developing kidney and, still later, filled with fluid, compressing and displacing the normal renal parenchyma found about it. The capsule about the cyst, its fusion with the kidney capsule laterally, and the presence of rudimentary glomeruli and compressed tubules within the capsule, along with a lack of evidence of communication with the renal pelvis, further substantiated this assumption."

Frazer (11), discussing the embryology of this portion of the kidney, believes that it is the failure of junction between the pelvic portion of the growing kidney and the metanephric blastema which may lead to the many varieties of congenital cystic kidney.

It is concluded that the case here reported does not represent a form of polycystic kidney, but that the cystic condition demonstrated represents a variation of the same embryologic abnormality.

A distinct fall in blood pressure from the sustained hypertension found on admission was noted following roentgen therapy, and this drop in blood pressure was maintained after operation up to the day of discharge from the hospital. No other instance of hypertension associated with congenital multilocular encapsulated serous cysts of the kidney has been noted, nor is any record made of the effect of roentgen rays on the blood pressure, or on the cyst, when these lesions are radiated. Mention is therefore directed to the blood pressure changes merely as an interesting phenomenon in the light of the present concept of the renal causes of hypertension. Further studies on the permanency of the relief obtained and its possible etiology are now in progress.

CONCLUSIONS

1. A unique case of congenital multilocular encapsulated serous cyst of the kidney occurring in a nineteen months old infant is presented and its embryology discussed.
2. A modification of Kampmeier's theory seems to offer the most reasonable explanation for its development.
3. There was an associated sustained hypertension present on admission but not evident at operation.
4. Attention is called to the difficulties encountered in making a clinical differential diagnosis between congenital multilocular encapsulated serous cysts of the kidney and renal embryoma (Wilms' tumor).

The authors are especially indebted to Mr. Adam Szczpinski of the University Hospital laboratory for his cooperation in the biochemical determinations made on the cyst fluid.

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VINETHENE ANESTHESIA*

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This paper is based upon the statistics gathered from a series of 100 consecutive tonsillectomies and adenoidectomies, four years' experience in the use of vinethene for myringotomies and other minor surgery in the office, three years of mastoidectomies, and one year's experience with the anesthetic agent used as an induction agent preceding ether for general surgery; also upon one case of acute appendicitis and three cases of acute mastoiditis in which vinethene was the only anesthetic administered.

Physical properties and pharmacology. Before beginning the discussion of this paper a review of the chemical, physical and pharmacologic properties of this anesthetic agent is given. Vinethene consists of about 96.5 per cent of pure vinyl ether, together with 3.5 per cent of absolute alcohol and .01 per cent of an oxidation inhibitor. The alcohol is added to prevent freezing of the moisture which accumulates on masks or in gas machines because of the cold induced by the high volatility of vinyl ether. The oxidation inhibitor is introduced to offset rapid decomposition on exposure to light and air.

Vinethene is a clear liquid dispensed in brown bottles. It has a characteristic odor, a low boiling point, 82 to 87 F., and decomposes when exposed to light or air. It should be kept in tightly stoppered bottles and stored in a cool dark place.

Advantages of vinethene. Some of the special characteristics worthy of mention are as follows:

1. Convenience, simplicity and ease of administration. A drop method was used and the anesthetic acted as pleasantly as nitrous oxide and ethylene.
2. The rapidity with which unconsciousness was produced.
3. Minimum excitement during the induction.
4. The speed with which the level of surgical anesthesia was reached.
5. Adequate degree of muscular relaxation which was attained.
6. The rapidity with which reflexes returned when the anesthetic was stopped.
7. The promptness with which complete consciousness was regained.
8. The rarity of vomiting and nausea.

Subjects and procedure. The patients ranged from nineteen months to sixteen years of age. More than 66 per cent of them were between the ages of four and eight years. The preoperative condition of these patients was satisfactory. Since the majority were in good health, only a small number showed evidence of malnutrition. .

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* Read before the Section on General Practice of Medicine and Surgery, State Medical Society, Pinehurst, N. C., May 19 to 21, 1941.

Premedication. Table 1 shows the type of medication used, which was worked out according to the age and the general state of nutrition of the patient. Atropine should be given in all cases to prevent excessive salivation, the same as for any general anesthetic. In myringotomies there is not a noticeable increase in saliva without atropine.

TABLE 1

AGE	DOSE
years	
2	$\frac{1}{400}$ grain atropine
3	$\frac{1}{300}$ grain atropine
4	$\frac{1}{200}$ grain atropine— $\frac{1}{2}$ grain seconal
$4\frac{1}{2}$	$\frac{1}{200}$ grain atropine— $\frac{3}{4}$ grain seconal
5	$\frac{1}{150}$ grain atropine— $\frac{3}{4}$ grain seconal
6	$\frac{1}{150}$ grain atropine— $\frac{3}{4}$ grain seconal
7	$\frac{1}{150}$ grain atropine—$1\frac{1}{2}$ grains seconal
8	$\frac{1}{150}$ grain atropine—$1\frac{1}{2}$ grains seconal
9	$\frac{1}{150}$ grain atropine—$1\frac{1}{2}$ grains seconal
10	$\frac{1}{150}$ grain atropine—$1\frac{1}{2}$ grains seconal
11	$\frac{1}{150}$ grain atropine—$1\frac{1}{2}$ grains seconal
12	$\frac{1}{150}$ grain atropine—$1\frac{1}{2}$ grains seconal—$\frac{1}{8}$ grain morphine
13	$\frac{1}{150}$ grain atropine—$1\frac{1}{2}$ grains seconal—$\frac{1}{8}$ grain morphine
14	$\frac{1}{150}$ grain atropine—$1\frac{1}{2}$ grains seconal—$\frac{1}{8}$ grain morphine
15	$\frac{1}{150}$ grain atropine—$1\frac{1}{2}$ grains seconal—$\frac{1}{8}$ grain morphine
16	$\frac{1}{150}$ grain atropine—$1\frac{1}{2}$ grains seconal—$\frac{1}{8}$ grain morphine

It was found to be unnecessary and inadvisable to give premedication to those patients who were to be ambulatory immediately after anesthesia, as in the cases of paracentesis of the ear drum and furuncle of the auditory canal. Premedication was also withheld in the shorter operations upon the eyelids and lacrimal ducts.

Method. Anesthesia was induced by the open drop technic, twelve to fourteen layers of gauze, approximately 8 by 8 inches, being placed over the face in such a manner that the area of gauze upon which the anesthetic was dropped did not come in direct contact with the skin or mucous membrane. Although mention is made of precautions against causing a burn or facial irritation, this was not experienced in the present series of cases. The use of a wire ether mask of the Yankauer type is suitable in older children and was used where vinethene was employed as the induction agent preceding ether anesthesia. Liquid albolene or mineral oil was applied as a protection to the eyes. The dropper tip was held close to the mask to prevent loss by evaporation in the air before the dropper reached the vaporization surface. The rate of administration was slow, between that of chloroform and ether; only 4 to 5 drops were administered during the first few breaths to prevent the sense of strangling or suffocation. This was gradually increased as the stage of unconsciousness approached. The patient was advised to count aloud, provided he could. Consciousness was lost in thirty to sixty seconds and usually there was no excitement manifested, except in the patients under four years of age who usually cried until going to sleep. The average time for the administration of surgical anesthesia was four and a half minutes. Surgical anesthesia is recognized by regular respiration, which is generally quiet and even, and relaxation of the skeletal muscles. The eyelids remain open and the eyeballs are active in the first plane. In the third plane anesthesia, the lids relax, the eyeballs are fixed, and the pupils usually dilate as in deep ethyl ether anesthesia. It requires from 60 to 90 drops per minute to maintain surgical anesthesia. An average of 100 cc. of vinethene is used per hour.

The amount of vinethene required varied from 15 to 50 cc., depending upon several factors such as prolonged bleeding, spongy tonsils, and the lack of proper sedation prior to the administration of the anesthetic. The average amount used was 26 cc. for each patient. Where more than two patients are to be operated on in sequence, it is more economical to start out with a 50 cc. bottle and supplement this with a smaller bottle. It may be of interest to note that vinethene is distributed in 10, 25, 50, and 75 cc. bottles.

The operating time, although somewhat variable, was also dependent upon the same factors which governed the amount of vinethene used, namely, prolonged bleeding, spongy tonsils, and the lack of preanesthetic sedation. One case was prolonged because of stoppage by a blood clot in the rubber tubing attached to the suction machine. The average operating time was eleven minutes. No patients were removed from the operating table until all bleeding had been checked. The operating time was considerably less, six to nine minutes, in the late cases of this series where there was no bleeding.

Difficulties of induction. No complications were encountered in this series. However, one patient, two years old, did stop breathing temporarily and became cyanotic after the operation was started. This was evidently a reflex condition caused by opening the mouth too wide and by too much flexion of the head. The child responded well to artificial respiration and oxygen.

Recovery. In all cases recovery was rapid and particularly free from untoward symptoms. Nausea and vomiting were conspicuous by their absence. These are among the outstanding characteristics of vinethene as an anesthetic. Absence of the excessive sweating so often seen with ethyl ether is also a salient point in its favor.

Vinethene as an induction agent. Vinethene as an induction agent was employed in all of the mastoid cases in this series, regardless of age, and was used solely in three cases of acute mastoiditis in patients of two months, eight months and eight years of age. The author has also used it routinely during the past year before ether anesthesia in general surgery and in one case of acute appendicitis in a patient of eight years of age. The boy who was operated upon for acute appendicitis and who had vinethene administered as the anesthetic agent was the son of a local surgeon. The choice between vinethene and ether was made in favor of the former because it was felt that vinethene would be less likely to produce bronchial irritation, since the patient already had a definite upper respiratory tract infection.

Vinethene has proved extremely satisfactory because of the simplicity of apparatus and administration and the smoothness and rapidity of anesthesia. It is of paramount advantage in situations in which a gas-oxygen induction is not available, as is the case in one local hospital. The psychologic factor of sparing the patient a prolonged ether induction is also important.

The premedication varied according to the ages of the respective patients. The usual drugs, atropine and morphine, and one of the barbituric acid preparations, nembutal or sodium amytal, in combination with the former or alone, were administered. The barbiturate derivative was given the night before and in the morning on which the surgery was performed. There were no cases of any excitement attributed to the anesthetic, and only one case of nausea and vomiting occurred after the administration of ether was started. No cyanosis was observed.

Postoperative course. From the hospital attendants and the surgeons in charge it was learned that nausea and vomiting occurred less frequently, the reaction time from the anesthetic was much shorter, and that postoperative gas pains and the tendency to gaseous distention of the bowels were greatly reduced as compared with straight ether anesthesia.

The young boy, eight years of age, developed a mild pneumonic infection on the second postoperative day, but this was more or less to be expected since he had a respiratory tract infection at the time of the operation.

CLINICAL OBSERVATIONS MADE BY DR. LEATH AND THE AUTHOR

1. A brief induction period was possible.
2. There was definitely less respiratory tract infection or irritation fol-

lowing tonsillectomies and adenoidectomies as compared to the cases that received ethyl ether. Vinethene was not adequate as an anesthetic for tonsillectomies on adult patients as they reacted too quickly and the pharyngeal reflexes were too active.

3. The excitement period was negligible.
4. There was considerable reduction in the number of cases of postoperative nausea and vomiting.
5. In the average patient the mental reaction was pleasant, with an occasional exception in adults.
6. There were no postoperative complications that could be traced to the anesthetic.
7. Sweating was absent.
8. The return to the conscious state was rapid.
9. There was a shorter duration of postoperative care. Ten to fifteen minutes were adequate for the nurse to be in attendance.
10. The smoothness of anesthesia was apparent.
11. The anesthetic was convenient to administer.
12. There was less mental torpor, as a result of inhaling the fumes, on the part of the anesthetist as compared with ethyl ether.
13. Less bleeding occurred during the operation.
14. There was less bleeding throughout the winter months as compared to the summer months. This should encourage the surgeon to do more tonsillectomies and adenoidectomies in cold weather.
15. The greater the preanesthetic sedation, the more tendency there was to prolonged bleeding.
16. There was no evidence of any skin or mucous membrane irritation with the use of gauze instead of a mask without the use of a face cream.

SUMMARY

From clinical experience in 100 consecutive administrations in tonsillectomies and adenoidectomies, perhaps an additional 100 administrations as an induction agent preliminary to ether, and four years' experience with this agent in the short cases that are seen in the office and home, the author feels that vinethene anesthesia has a definite place in the surgical armamentarium. Vinethene serves as an unexcelled general anesthetic agent for office and outpatient or home practice.

PORENCEPHALY*†

CHARLES W. REAVIS, M.D. AND WALTER L. KILBY, M.D.

BALTIMORE, MD.

Porencephaly is a relatively rare condition, but one with which every radiologist should be familiar. In reviewing a series of 182 consecutive encephalograms taken over a six year period this condition was encountered only twice, once in an infant and once in an adult. Recently a case has been seen in an infant discovered by ventriculography. Previous to the general use of pneumo-encephalography, the abnormality was one mainly diagnosed at the autopsy table and a large portion of the literature deals with it from the pathologic point of view. According to Patten, Grant, and Yoskin (12), this condition is a neurologic entity, with many varied clinical manifestations. The diagnosis can be made clinically.

The term *porencephaly* is derived from the Greek and means *pore brain.* It was first used by Heschl (5) in 1859 to describe a condition occurring in eight cases in which there was a defect in the brain, with cavity formation within the cerebral substance. He thought that it was caused by maldevelopment. However, the condition itself was known before this description by Heschl.

Definitions of the term porencephaly vary in detail, but all recognize a communicating defect of the brain with the ventricular system. Patten et al. (12) use the term as set forth by Lecount and Semerak (11) to "signify a defect in the cerebral or cerebellar structure appearing as a cystlike cavity communicating with the ventricles or separated from them only by a thin layer of brain tissue, covered on the outside by the pia-arachnoid and filled with a clear, colorless fluid." They believe that its use should be based on anatomic structures rather than etiology. Laird (10) interprets it to mean "any brain presenting a cerebral cortical defect covered by arachnoid and communicating or nearly communicating with either or both lateral ventricles." Jaffe (6) writes that it is usually defined as "a funnel-shaped defect of the brain which extends from the surface of the hemisphere close to or into the lateral ventricle. The defect is covered by a membrane of varying thickness and is filled with clear, colorless fluid. Thus, on external examination it appears as a cyst."

Pathologically, the lesion is one of a cystlike cavity within the brain substance, filled with fluid and covered by a firm thin membrane composed of dense fibrillary glia. The pyramidal cells of the cortex about the lesion are deranged and are fewer in number than in other regions. The cells are

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* From the Department of Roentgenology, School of Medicine, University of Maryland.

† Received for publication November 24, 1941.

pale, and the cell body may be hard to outline. The nuclei are pale staining and tend to be disintegrated. Hydropic and swollen cells are encountered. There is an excess of the glial cells and fibrils in this portion of the cortex. There may be vacuolization of the nervous tissue, which increases as it approaches the cyst area. Other changes depend upon the origin of the defect. There may be evidence of old or recent hemorrhages present. If the defect is large and involves any of the important centers, then frequently there is atrophy or agenesis in remote parts of the brain. Reeves (14) reports two cases of porencephaly associated with agenesis of the corpus callosum and is of the opinion that this association is of frequent occurrence and often overlooked. Lecount and Semerak (11) also report a case with agenesis of the corpus callosum.

Following the introduction of the term porencephaly, Heschl (5) later included those changes of a regressive destructive nature caused by vascular occlusion, as well as those resulting from maldevelopment. Later, various authors added other etiologic agents. Kundrat (9) included any defect which had the arachnoid as a covering. Those defects caused by circulatory disturbances soon were accepted by other writers. Numerous other theories have been advanced until at the present time there still remains some controversy as to just what causative conditions the term should include. Jaffe (6) feels that trauma is possibly the greatest single etiologic agent. Globus (4) advocates placing it instead on an inflammatory basis. He gives in detail a case report of a microcephalic with marked atrophy and diffuse sclerosis, which began as meningo-encephalitis and ultimately led to occlusion of the blood vessels with a cortical defect. Wiglesworth (16) presents a case in which it was definitely of intra-uterine origin and occurred at about the sixth week following an arrest of cortical development in the telencephalic wall.

The literature from an etiologic basis has been well reviewed recently by Lecount and Semerak and also by Jaffe. The majority of the writing on this abnormality has appeared in foreign journals. It is generally agreed that the condition may be congenital or acquired. Circulatory disturbances, hemorrhage, embolism, thrombosis, infections, trauma, and congenital malformations play a part. Laird (10) offers a classification as to the etiology on a plasmic and somatic basis, the former dealing with any influence upon the germ plasma before fertilization and the latter after fertilization. He divides the somatic basis into three groups: the congenital, the natal, and the acquired. The congenital group includes any influences acting during intra-uterine life, such as cramplike contractions of the uterus, prenatal encephalitis, or other infections and anomalies of the placenta. Natal causes are prolonged labor and birth trauma. These cause circulatory changes which may result in necrosis associated with infection. The acquired causes include those occurring later in life. A single factor or any combination of factors may cause the occurrence of a porencephalic cyst.

There is no definite symptomatology associated with porencephaly. It may exist asymptomatically and be found postmortem, and it is not incompatible with life. One of the most striking features is the wide gap between the symptomatology and the amount of brain tissue actually destroyed. Marked disturbances in sensation and function may be present or only a few changes may be noted, depending upon the site and extent of the lesion. Some occur in the so-called silent areas of the brain, thus producing no symptoms at all. Patten et al. (12) studied a series of nine children who had convulsive seizures as the predominating symptom. Bernstein (1), in a series of seven cases out of 1000 admissions to the Psychiatric Clinic of the Yale School of Medicine, found all to have convulsions or some psychiatric disturbance. The seven came from tainted stock and were of less than average intelligence. Many had the so-called epileptic constitutionality and showed abnormal personality traits. Mental deficiency or epilepsy are practically always found in association with porencephaly. In a review of many case reports from the literature it is noted that almost any neurologic symptom or sign may be present, according to the location of the lesion. One side of the body may be less developed than the other. Severe motor dysfunction, and spasticity or sensory dysfunction may be observed. Other defects elsewhere in the brain or even in remote parts of the body may occur. Thus, there is no definite clinical picture, the condition occurring in any age or in either sex.

Very little attention has been paid to the roentgenologic aspect in the diagnosis of this condition, and the roentgenologic literature is conspicuous for its scarcity. A diagnosis of porencephaly cannot be made from the conventional roentgenogram of the skull, but certain features may call attention to an abnormality. There may be slight asymmetry in the size of the calvarium or in the thickness of the bones. The smaller side may be a manifestation of an atrophic brain, and the thickness of the affected side may be compensatory hypertrophy. Localized areas of decreased density may be present, indicating bone atrophy from the pressure of the porencephalic cyst when it is in immediate proximity to the bone. Hypertrophy of the air sinuses of the affected side from increased pneumatization should suggest the presence of an atrophic lesion on this side. It must be remembered that these changes are those which go along with any condition causing atrophy of the cerebral cortex, and none are pathognomonic of porencephaly. Cysts of the leptomeninges or any space-occupying lesion adjacent to the bone may cause localized areas of bone atrophy. Hemiatrophy and hemihypertrophy are common following trauma during infancy and childhood. However, this condition is not limited to those atrophic manifestations as it also may be seen in the large skull of the hydrocephalic.

There may be no change whatsoever demonstrable in the skull. This is well shown in those cases in which trauma occurs during adult life, and as a result of necrosis the defect in the brain occurs.

The roentgenologic diagnosis of porencephaly is made from the pneumoencephalogram, the gas having been injected either directly into the ventricles or by the spinal route. Roentgenologically, any defect communicating with a ventricle and appearing in the tissues of the brain may be considered a porencephaly. This may be an area of cerebral necrosis, or any loss of brain substance or cyst-like area which communicates with the ventricles and thus may be filled with air to render the defect visible.

The usual finding is that of a single cavity above the level of the tentorium. The cavity may be of any size or shape and may occur anywhere within the brain. Jaffe (6) believes that it most frequently occurs in the parietal region about the central convolution. It is well defined and circumscribed, and a communication with one of the lateral ventricles can generally be demonstrated. However, the cavities may be multiple, bilateral or loculated. If the ventricular system is not well filled, a roentgenogram in the erect position may reveal a fluid level with the gas above it. Attention has been called by Pendergrass and Hodges (13) to the value of a twenty-four hour examination in this condition, with the idea that fluid may remain in the cavity at the original study and during the interim pass out and be replaced by the gas, thus being visualized at the later examination. The basal cisterns are often well visualized and may be slightly enlarged.

Porencephaly should not be confused with cerebral atrophy, though it occurs as a manifestation of this condition. The massive subarachnoid space over the cortex so frequently seen is often present and the differentiation of the porus may be difficult. An internal hydrocephalus is generally noted. This is also a part of the atrophic process and varies with the degree manifested. The homolateral ventricle may be the smaller and may be displaced to the affected side because of the atrophy; however, if an arachnoiditis is associated or has been present, the brain may be adherent and no cortical air demonstrated. After trauma the porencephalic cyst may be the only demonstrable abnormality.

The differential diagnosis includes an incompletely filled dilated ventricle, a gliomatous, hydatid or other similar cyst, and a subdural hematoma. If a dilated ventricle is incompletely filled, it may be difficult to distinguish between this and the porencephalic cavity. By careful study the course of the ventricle will usually be detected. A gliomatous cyst causes no atrophy of the brain and the ventricular shift is to the opposite side. A pressure defect may be demonstrated upon the ventricle. If air is being introduced by the direct route, the needle may be inserted into the cyst, the contents withdrawn, and its margins outlined with air. If this air then goes into the ventricular system, the communication of the porencephaly may be demonstrated. The gliomatous cysts generally do not communicate with the ventricles.

The history of injury and persistent headache in the subdural hematoma usually will cause this condition to be suspected. There may be a pressure encroachment on the ventricle of the affected side. The ventricular shift is laterally away from the lesion and site of injury. If the pineal gland is calcified, there is a lateral shift to the opposite side. Gas confined between the inner membrane of the clot and the collapsed brain generally extends laterally and ventricularly from the midline. This sign is pathognomonic of subdural hematoma. Such an air space should not be confused with porencephaly.

CASE REPORTS

Figure 1 shows the encephalograms of a white female infant who was admitted at the age of eight months with the complaint of improper development, enlarged head, and muscular weakness. The infant had been throwing the head back and manifested signs of muscular weakness in the neck. There was excessive motion of the hands, and the tongue protruded from the mouth. Delivery of the patient had been normal and she was breast-fed for one month, after which time cow's milk and dextrimaltose were prescribed. At birth her weight was seven and one-half pounds and at eight months it was twelve pounds. There had been no convulsions nor serious illnesses, only mild grippe at one month. The family history was negative, except that the mother had "hysterical nervousness." Two brothers and one sister were living and well. At an early age the child took no notice of things about her and seemed to be retarded.

Physical examination revealed an enlarged head, with symptoms of mental and physical retardation. The eyes were turned upward most of the time and the tongue protruded. There was no cervical rigidity nor weakness of the extremities. Spinal fluid serology was negative; two white blood cells and many red blood cells were found. The blood pressure was 100 mm. of mercury systolic and 50 mm. of mercury diastolic. Blood and urine examinations were negative. A ventriculogram showed normal lateral and third ventricles, with cortical atrophy.

The child was discharged several days later with a diagnosis of internal hydrocephalus, with cortical atrophy and mental retardation, both unimproved. She was readmitted at the age of fourteen months with the same complaints, plus the inability to walk or hold up her head. There had been no appearance of teeth. The physical and laboratory findings were not unusual, but an encephalogram after the injection of 110 cc. of air revealed an area in the right parietofrontal region filled with air and communicating with the right lateral ventricle. This area measured 13 by 20 by 25 mm. and represented a porencephalus. There was also dilatation of the ventricular system and the basal cisterns, and considerable air was noted over the cortex. The anterior fontanel was slightly widened, indicating a retardation of bony development.

In this case there is a great possibility that the defect was congenital in nature, because it or some similar factor was present since birth and caused a lack of normal development in this infant.

Figure 2 shows the encephalograms of a white male, age 53, who was admitted with the complaint of a "buzzing in the head," with numbness and tingling of the left side. Six months previously he suffered a left hemiplegia, which occurred at night while at rest, the patient awakening and finding the left side paralyzed. Previous to this time he had been healthy, and four months before had a systolic blood pressure of 122 mm. of mercury. Since the hemiplegia he had two attacks of tremor and rigidity of the left arm and leg, with drawing of the head to the left. However, of late he had regained some use of his left side.

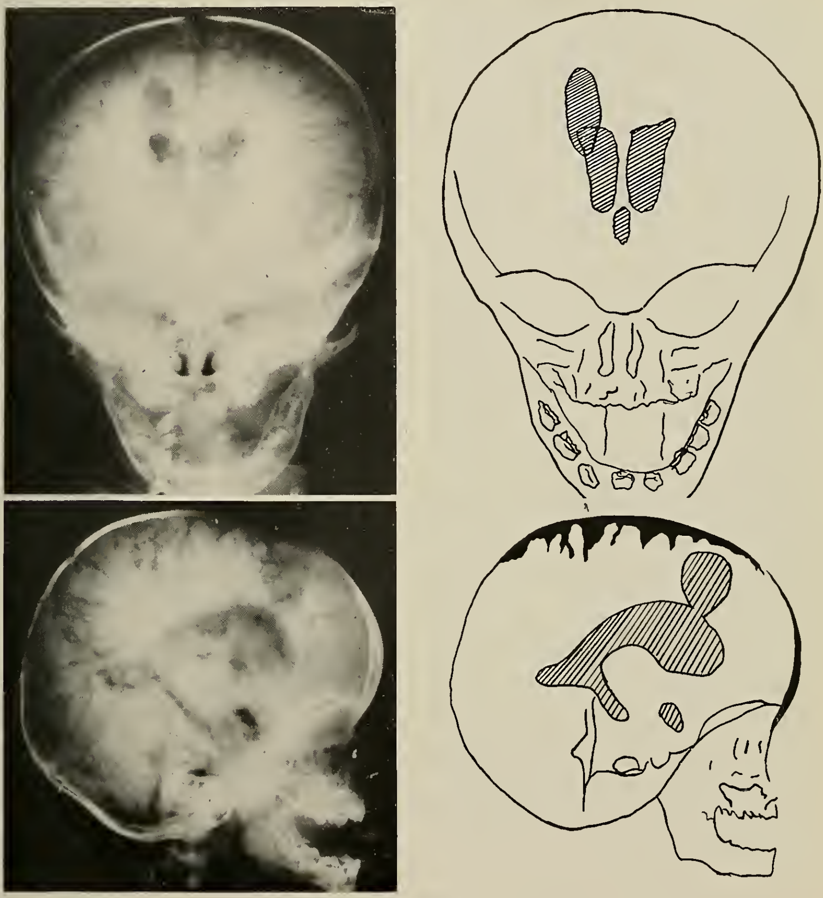

FIG. 1. Encephalogram of a fourteen months old female showing a porencephalic defect in the right cerebrum, probably congenital in origin.

At the time of admission his blood pressure was 150 mm. of mercury systolic and 100 mm. of mercury diastolic. He was well nourished and developed. There was definite weakness of the left upper and lower extremities; hyperactive deep reflexes were present on the left; and there was a left ankle clonus and a suggestive left plantar extensor response. Roentgen examination of the skull was negative. Routine laboratory examinations were negative, except for a trace of albumin in the urine. The spinal fluid was clear. Serology of the spinal fluid was negative, its pressure being 134 mm. of water Encephalograms revealed an area measuring 14 by 30 by 30 mm. in the right parieta region which was filled with air and communicated with the right lateral ventricle. There was also dilatation of the ventricular system, but no shifting in any direction (Fig. 2).

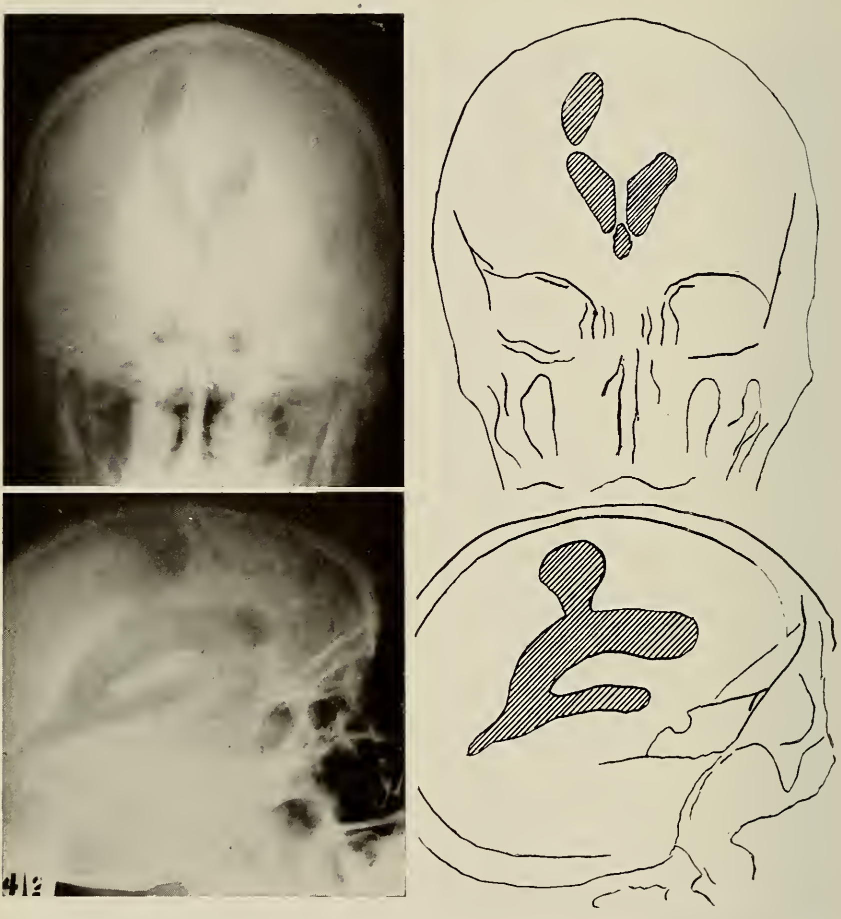

Fig. 2. Encephalogram of fifty-three year old male clerk with a porencephalic defect in the right parietal region, circulatory in nature.

This defect represented a porencephalus of circulatory origin occurring in an adult, probably caused by a localized hemorrhage, with resultant softening and necrosis leading to a defect in the cerebral hemisphere.

The treatment of porencephaly is limited generally to social readjustment, psychotherapy, and psychiatric measures. Surgical intervention may be of definite value according to Patten et al. Of six patients out of eight surviving operation, three had complete relief from convulsions and three were greatly improved. The prognosis varies according to the extent of the lesion and its location, but little can be hoped for in the way of overcoming the deficiency if such be present.

SUMMARY

A resumé of the condition known as porencephaly is presented, with special reference to the roentgenologic aspects. It is illustrated by two case reports, with the idea of bringing this condition to the attention of physicians, and to stress the fact that the condition can be diagnosed during life with little difficulty by means of pneumo-encephalography.

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ONE HUNDRED AND FOURTEENTH PROGRAM MEETING

The One Hundred and Fourteenth Program Meeting was held on Tuesday, October 21, 1941 in Gordon Wilson Memorial Hall. The following program was presented:

"The Relationship of Chemical Structure to Fungicidal Activity," by Mr. Richard H. Harry, Departments of Pharmaceutical Chemistry and Bacteriology, School of Pharmacy, University of Maryland.

"The Porphyrin Fluorescence of Harderian Glands in Mice of Cancer-Susceptible and Cancer-Resistant Strains," by Drs. F. H. J. Figge and L. C. Strong, Departments of Anatomy, School of Medicine, University of Maryland, Baltimore, and Yale University School of Medicine, New Haven, Connecticut.

Abstracts of these papers follow.

THE RELATIONSHIP OF CHEMICAL STRUCTURE TO FUNGICIDAL ACTIVITY

R. H. Barry, B.S., M.S.* †

BALTIMORE, MD.

The introduction of alkyl, halogen or carboxyl groups into phenol increases the fungicidal activity of the phenol. O-alkyl derivatives of p-halogenated phenols are more fungicidal than p-alkyl derivatives of o-halogenated phenols. Straight chain alkyl substituted phenols are more active than branched or cyclo-alkyl phenols. Carboxyl introduced ortho added to the phenolic hydroxyl exerts more fungicidal activity than when substituted meta or para. A mixture of benzoic and salicylic acids is more active against "ringworm fungi" than either one used alone. The nitro group in phenolic compounds does not increase the fungicidal power significantly. The introduction of a second hydroxyl group into phenol lowers the fungicidal activity of phenol. The introduction of alkyl g groups into resorcinol helps to compensate for the lowered fungicidal activity. Resorcinol mono ethers with aliphatic or aromatic substituents show practically the same fungicidal power as when the substituent is attached to the nucleus. The thio-alkyl ethers of phenol show fungicidal activity comparable to the alkyl phenols.

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* From the Departments of Pharmaceutical Chemistry and Bacteriology, School of Pharmacy, University of Maryland.

† Read at the meeting of the University of Maryland Biological Society, October 21, 1941.

The phenylmercuric halides are highly toxic for fungi. Halogen substituted para to the mercuri-halide group (HgX) is more active than ortho or meta substitution.

Recent work indicates that the chlorothymolmercuric halides show promise as fungicides for *E. interdigitale* and *A. gypseum.*

The quaternary compound alkyl dimethylbenzyl ammonium chloride has been reported a good fungicide, especially in the presence of serum.

The triphenylmethane dyes are more fungicidal than those of the phthalein and azo classes.

THE PORPHYRIN FLUORESCENCE OF HARDERIAN GLANDS IN MICE OF CANCER-SUSCEPTIBLE AND CANCER-RESISTANT STRAINS* †

FRANK H. J. FIGGE, PH.D.‡ AND LEONELL C. STRONG, PH.D.§

BALTIMORE, MD. AND NEW HAVEN, CONN.

Mammary cancer susceptibility in mice and fluorescent phenomena associated with vitamin deficiency in rats were being investigated simultaneously by one of the authors. This led to the observation that there was a marked variability in the red fluorescence of the harderian glands of mice. The first observations were made on mice over 300 days old and included animals of only two strains. The harderian glands of old C_3H (mammary cancer-susceptible) mice were found to exhibit a marked red fluorescence in ultraviolet light. In the glands removed from old mice of the JK (mammary cancer-resistant) strain no red fluorescence was observed. Later the harderian glands of over 750 mice, from thirteen inbred strains and three hybrid strains, were examined for red fluorescence. The degree of variability was expressed by the symbols 0, +, ++, +++, ++++. The two extreme variants were found in adult mice beyond 300 days of life of the JK cancer-resistant strain (0–+) and mice of the C_3H cancer-susceptible strain (+++–++++). Mice from strains showing intermediate susceptibility exhibited intermediate degrees of fluorescence. An age variation was observed. Before the eyes were open at fourteen days no fluorescence of orbital contents was detected. In early sexual maturity a high fluorescence was seen. This was found to decrease in intensity in JK mice with advancing age and was completely absent in old mice. This decrease in fluorescence with advancing age was not observed in C_3H mice.

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* This investigation was carried out at Yale University. It was aided by grants from the Jane Coffin Childs Memorial Fund for Medical Research and The Anna Fuller Fund.

† Read at the meeting of the University of Maryland Biological Society, October 21, 1941.

‡ Department of Anatomy, School of Medicine, University of Maryland. Rockefeller Fellow, 1940–41, Yale University School of Medicine.

§ Department of Anatomy, Yale University School of Medicine.

Red fluorescence of the harderian gland is an indication of the presence of porphyrins. There is some evidence to indicate that porphyrins may be involved in the synthesis of hemoglobin. Porphyrins are also known to be important constituent parts of catalase, the Pasteur enzyme, and cytochrome C. The fact that there is a close parallelism between the degree of red fluorescence of the harderian gland and the degree of cancer susceptibility in such a number of strains of mice indicates a relationship between cancer susceptibility and the red fluorescence of harderian glands. The red fluorescent porphyrin in the harderian gland may be an indicator of a more obscure physiologic process related to cancer susceptibility.

ONE HUNDRED AND FIFTEENTH PROGRAM MEETING

The One Hundred and Fifteenth Program Meeting was held on Tuesday, November 18, 1941 in Gordon Wilson Memorial Hall. The program consisted of the following papers:

"Quinine and the Count of Chinchon," by Miss Georgianna S. Gittinger, Department of Pharmacology, School of Pharmacy, University of Maryland.

"Metabolism and Toxicity Studies with Mannitol and Sorbitol in Man and Animals," by Dr. Fred W. Ellis, Department of Pharmacology, School of Medicine, University of Maryland.

Abstracts of these papers follow.

QUININE AND THE COUNT OF CHINCHON

Georgianna S. Gittinger, A.B., M.A.* †

BALTIMORE, MD.

The official record of each Viceroy of Peru in the colonial period was known as the Diario de Lima. From the Diary of Suardo, secretary to the Count of Chinchon, Viceroy from 1629 to 1640, is derived a detailed story of the introduction of quinine into European medicine; and also the little known fact that it was the Count and not his Countess who suffered from malaria and was cured by the bark.

METABOLIC AND TOXICITY STUDIES WITH MANNITOL AND SORBITOL IN MAN AND ANIMALS

Fred W. Ellis, Ph.D.‡ §

BALTIMORE, MD.

Mannitol and sorbitol belong to a group of compounds which are chemically related to the simple sugars. They possess an OH group for each carbon atom in the molecule and, therefore, are called sugar alcohols. Although mannitol and sorbitol are not true sugars, the close resemblance between the chemical structures of these substances and dextrose is of sufficient significance and interest to warrant their metabolic study.

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* From the Department of Pharmacology, School of Pharmacy, University of Maryland.

† Read at the meeting of the University of Maryland Biological Society, November 18, 1941.

‡ Research Fellow in Pharmacology, School of Medicine, University of Maryland.

§ Read at the meeting of the University of Maryland Biological Society, November 18, 1941.

These compounds, when ingested in sufficient amounts, produce a laxative action which limits nutritional studies in mice and rats and interferes to some extent with quantitative metabolic studies in man. Owing to the fact that the laxative effect of mannitol is much greater than that of sorbitol, the latter can be tolerated by man and animals better than the former.

When given by stomach tube to fasted monkeys, sorbitol serves as a better precursor of liver glycogen than mannitol. In fasting human subjects, sorbitol has the capacity to increase the respiratory quotient without appreciably elevating the blood sugar. After the oral administration of 25 or 50 gm. of sorbitol the respiratory quotient rises within two hours as high as after an equal quantity of dextrose, but the blood-sugar level does not change significantly. Mannitol does not produce a notable effect on either the respiratory quotient or the blood sugar under similar conditions. It is suggested that the possible intermediate metabolism of sorbitol is probably dependent upon its conversion within the body to levulose.

The daily feeding of mannitol and sorbitol to monkeys over a period of three months produced no histopathologic findings or toxicologic indications. In man the daily ingestion of these compounds for one month caused no significant changes in the red blood cell count, the nonprotein nitrogen, or the carbon dioxide combining power of the blood. The phenolsulfonephthalein test indicated no kidney damage.

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An official transcript of the records of courses attended in college should also be sent by the college registrar directly to the Committee on Admissions.

Applicants may be requested to appear for interviews. An application fee of $2.00 must accompany all applications. The application and all pertinent credentials must be filed no later than September 15 of each year.

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Matriculation fee (paid once)	10.00
Tuition fee (payable in $25.00 installments at the beginning of each semester)	50.00
Laboratory fee	35.00
Total	$97.00
Second year	
Tuition fee (payable as above)	50.00
Laboratory fee	15.00
Graduation fee	5.00
Total	$70.00

Course of Study

The course of study will extend over a period of two school years and a portion of the intervening summer—a total of twenty months. Students will be required to take the scheduled courses in biochemistry, bacteriology and immunology, and clinical pathology in the School of Medicine. The remaining time will be spent in supervised work in the laboratories of the University Hospital and the Department of Pathology of the School of Medicine. The divisions of the hospital laboratory in which students will work are: Bacteriology, serology, clinical chemistry, hematology, electrocardiography and basal metabolism, and in histologic technique in the Department of Pathology of the School of Medicine.

Certificate of Graduation

A certificate of graduation will be granted by the School of Medicine of the University of Maryland upon successful completion of the course.

ITEMS

THE DOCTOR JULIUS FRIEDENWALD MEMORIAL LECTURE

As a fitting tribute and memorial to the late Doctor Julius Friedenwald, a lectureship has been established at the School of Medicine of the University of Maryland by contributions from his friends. Mrs. Julius Friedenwald has contributed a sufficient sum to the fund to insure a perpetual series of lectures.

It was impossible in the short time in which this lectureship was created to approach all of the many friends of this prominent physician. Anyone wishing to contribute to this fund may send his check to "The Doctor Julius Friedenwald Memorial Lecture Fund" in care of Dr. Maurice Feldman, 2425 Eutaw Place, Baltimore, Maryland.

The following is a list of the sponsors and contributors:

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THE BRESSLER ALUMNI RESEARCH CAMPAIGN

The following are additional subscribers to the Bressler Alumni Research Fund:

Dr. Max Trubek	New York City
Dr. and Mrs. William L. Fearing	Baltimore, Md.
Dr. D. J. Pessagno	Baltimore, Md.
Dr. Frank J. Schwartz	Spring Valley, N. Y.
Dr. Samuel Zeiger	Brooklyn, N. Y.

Dr. Thomas B. Aycock, Professor of Clinical Surgery, delivered an address on "Surgical Treatment of Gastric and Duodenal Gastric Ulcers" at the sectional meeting of the Panhandle Division of the West Virginia Medical Society on December 10, 1941 at Martinsburg, West Virginia.

The Annual deSchweinitz Lecture, Section of Ophthalmology of the College of Physicians of Philadelphia, was delivered on November 19, 1941 at the College by Dr. Harry Friedenwald, Professor of Ophthalmology, Emeritus. There was a dinner in honor of Dr. Friedenwald at the University Club before the lecture.

Dr. George Silverton, Class of 1932, has been commissioned First Lieutenant with the medical detachment of the 176th Infantry, 29th Division, and is stationed at Fort Bragg, North Carolina.

Dr. Louis A. M. Krause, Associate Professor of Medicine, has completed five lectures in the series of eleven he is giving in Chemical Hall at the University. These talks are not only educational but highly interesting. They are open to physicians, students and the general public without charge. The remaining lectures will be given at 5:00 p.m. on February 3, 10, 17, and 24, and March 3 and 10, 1942.

Dr. Joseph C. Evans, Class of 1903, San Pedro, California, visited the University Hospital and School of Medicine on September 23, 1941.

Dr. William S. Love, Jr., Associate Professor of Medicine and Instructor in Pathology, spoke on "Cardiac Emergencies" at the meeting of the Maryland Academy of Medicine and Surgery held on November 18, 1941.

Dr. Stuart D. Sunday, Class of 1936, has been commissioned First Lieutenant in the Army Medical Corps and is stationed at Trinidad, British West Indies.

Dr. John R. Agnew, Class of 1914, has been assigned to active duty as medical examiner for the Marine Corps, United States Naval Reserves. Dr. Agnew was formerly senior attending surgeon at the Wesson Memorial Hospital and attending surgeon at the Health Department Hospital of Springfield, Massachusetts.

Dr. Charles M. D'Alessio, Class of 1937, has opened an office at 18 Elizabeth Street, Derby, Connecticut, for the general practice of medicine and surgery.

Dr. J. S. Miranda, Class of 1908, of Havana, Cuba, recently paid a visit to the School of Medicine.

Dr. E. N. Boccanegra-Lopez, Class of 1916, has opened a new office at 875 Fifth Avenue, New York City. His practice will be limited to dermatology.

Dr. W. C. Arthur, Class of 1897, was retired on service pension by the B. F. Goodrich Company of Akron, Ohio on November 1, 1941, after serving since February 1, 1920 as head of this firm's surgical dispensary. Dr. Arthur is now living in Ellwood City, Pennsylvania.

MEDICAL LIBRARY NOTES

EXHIBITS

Exhibits of rare and historical materials illustrating the development of medicine have come to play an increasingly important part in medical education. It is a distinct pleasure, therefore, to report that the acquisition of two exhibit cases has made it possible for the Medical Library to inaugurate a program of exhibits designed not only to foster acquaintance with the many unusual items in the collection, but also to emphasize books which have been landmarks in medical history. The first two exhibits held during the months of September, October and November were entitled "Military and Naval Medicine and Surgery" and "Anatomy through Five Centuries," respectively. The schedule for the remainder of the year follows: December—"Contributions to Physiology"; January—"The Early Pharmacists and Chemists"; February—"Pathology from the Beginning"; March—"The Great Clinicians"; April—"The History of Surgery and Anesthesia"; May—"Obstetrics"; June—"Contributions to Medicine by the University of Maryland School of Medicine and College of Physicians and Surgeons." Suggestions for exhibits will be received gladly.

THE RETURN OF THE PRODIGAL

An interesting story is connected with Volume 2 of Fourcroy's "Chemistry" (1786) now in the Crawford Collection. This particular volume recently came into the hands of Dr. W. W. Francis of McGill University. On noticing Dr. Crawford's bookplate in the volume he wrote to inquire if that particular one was missing from the Library's set. Upon receiving word in the affirmative Dr. Francis kindly made arrangements for the book to be presented to the Medical Library. It is thought that this particular volume must have been loaned to someone by Dr. Crawford before his death in 1813 and thus was not included in the group of books purchased from his estate to form the nucleus of the Medical Library. After more than one hundred years of wandering this little book returns to its proper position in the set!

HOURS

A careful count kept over a period of two years showed that few patrons used the Library after 7:00 p.m. In view of the understaffed condition of the Library, the Medical Library Committee recommended and the Medical Council approved the following schedule of hours effective October 1, 1941.

Monday through Friday—9:00 a.m. to 7:00 p.m.; Saturday—9:00 a.m. to 4:00 p.m.

1940–41 IN BRIEF

The number of volumes in the Library on October 1, 1941 was 21,261; 597 volumes were added during the year; about 350 copies of Journals and Transactions were received; 5607 books were borrowed for home use, as compared to 5438 in 1939–1940.

REUNION

CLASS OF 1921 OF THE SCHOOL OF MEDICINE, UNIVERSITY OF MARYLAND

The Class of 1921 held its twentieth reunion on June 4 and 5, 1941. The festivities started at the Lord Baltimore Hotel in the morning and afternoon of June 4. At night thirty-eight members attended the class banquet. Twenty-three members of the Faculty were present, but some could not attend because of other commitments. Those present were:

Faculty: Drs. Arthur M. Shipley, Harry Friedenwald, William S. Gardner, H. Boyd Wylie, H. L. Sinskey, R. W. Locher, Albertus Cotton, C. Reid Edwards, J. Dawson Reeder, Joseph Gichner, Irving J. Spear, Edgar Friedenwald, Harvey G. Beck, George M. Settle, Edward A. Looper, C. L. Joslin, W. H. Toulson, L. J. Rosenthal, Frank J. Kirby, Charles Bagley, Jr., George A. Strauss, Mr. Edward Smith, and the late Dr. J. W. Downey.

Class Members: Drs. Francis L. Bagli, Baltimore; Bruce Barnes, Seaford, Del.; Carl Fisher Benson, Baltimore; John Ralph Bernardo, Bristol, R. I.; Samuel Hearn Culver, Baltimore; Herman J. Dorf, Baltimore; Charles F. Fisher, Clarksburg, W. Va.; Daniel S. Fisher, LaPlata, Md.; Charles J. Foley, Havre de Grace, Md.; Joseph P. Franklin, Cumberland, Md.; Leon Freedom, Baltimore; John Stanley Grabill, Mt. Airy, Md.; Cyrus Eugene Hawks, Rockville, Md.; Julius D. Holly, Baltimore; Albert Jaffe, Baltimore; Vincent Joska, Baltimore; George R. Joyner, Suffolk, Va.; Daniel F. Keegan, Bridgeport, Conn.; Richard Kemp, Washington, D. C.; Benjamin Luban, Newark, N. J.; Arley McCoy, Elm Grove, W. Va.; Stanley W. Matthews, Fort Benning, Ga.; Lyle J. Millan, Baltimore; Thomas R. O'Rourk, Baltimore; Anthony Pacienza, Baltimore; Harold C. Pillsbury, Baltimore; Ralph Johnson Plyler, Salisbury, N. C.; Joseph Pokorny, Baltimore; Ferdinand A. Ries, Sykesville, Md.; James Barry Ryon, Terre Haute, Ind.; Fred C. Sabin, Little Falls, N. Y.; Jesmond Schilling, Erie, Pa.; Solomon Sherman, Baltimore; George E. Shannon, Baltimore; Louis M. Timko, Cleveland, O.; George Wells, Baltimore; J. Herbert Wilkerson, Baltimore; Mortimer H. Williams, Roanoke, Va.

On the following day the class went to the alumni luncheon at the University Hospital and inspected the new Bressler Building. That afternoon Dr. and Mrs. Leon Freedom entertained at their home with a delightful cocktail party, and at night the class attended the Annual Banquet of the Medical Alumni Association. As a result of some speeches made by members of the class and Faculty, the class representatives will endeavor to create a scholarship endowment fund at the University of Maryland in memory of the deceased members of the Class of 1921. The reunion ended on June 6 and all those present returned to their homes. Everyone will look forward to the next reunion in 1946.

Jesse Wright Downey, Jr.

OBITUARIES

JESSE WRIGHT DOWNEY, JR., M.D.

Dr. Jesse Wright Downey, Jr. died on November 18, 1941. He taught otology at this University for many years, as Associate in 1916, as Clinical Professor from 1917 to 1928, and as Professor from 1929 until his death.

Dr. Downey was born in New Market, Frederick County, Maryland on August 31, 1882. His father was Dr. Jesse Wright Downey, a country physician of the finest type, sturdy, wise, sympathetic and beloved. The son followed his father in profession and in quality. After a course at Frederick College he entered the medical department of the University of Virginia, where he was graduated in 1905. He came to Baltimore and was appointed an intern at Mercy Hospital. After a year's service he became a resident physician at the Baltimore Eye, Ear and Throat Hospital. His connection with this hospital continued until his death; he rose from assistant to assistant surgeon, and then to surgeon, and took a leading part in the affairs of the hospital as secretary of the Board of Trustees and as chairman of the Surgical Staff. He was a member of the Medical and Chirurgical Faculty, of the Southern Medical Association, and of the American Otolological Society.

These bald facts indicate the public activities of a man who came to Baltimore as a young graduate in medicine and who rose to an honorable position in the estimate of his professional colleagues and of the public. They confirm what is well known: that he was an earnest student, an able practitioner, a skillful surgeon, a proficient teacher, deeply interested in his chosen field and in his classes of medical students. However, they fail to reflect his character and it is this that it is most difficult to sketch clearly and adequately. The fine qualities that were blended in what we call his personality were many and attractive. There was an earnestness and sincerity in his attitude to what he looked upon as his duties,—personal, civic and professional; there was true cordiality in his relations with those close to him, with his professional colleagues and with his friends. But this earnestness and sincerity were enlivened by a youthful, sprightly and social spirit. He was fond of the finer things in life, in nature, in literature and in humane interests. He was loyal to his friends, as he was devoted to those near him.

Dr. Downey was heavily stricken by deep sorrows but, though frail of build, he bore them with that great strength which his faith and his sense of courageous duty lent him; in this he had the unwavering support of a devoted helpmate whom he had married in 1914.

Dr. Downey's charm lay in the gentle, cheerful, friendly, cultivated gentleman he was.

HARRY FRIEDENWALD, M.D.

WILLIAM HENRY MARSH, M.D.

Dr. William Henry Marsh, U. of Md. 1876, aged 90, died on November 12, 1941 at the United States Marine Hospital in Baltimore.

Dr. Marsh was born in Baltimore on September 14, 1851. Following his graduation he served for two years as senior resident physician at Bay View Asylum, now a part of City Hospitals, before entering into practice at Solomon's Island. In addition to a long and active career here he also served with the United States Marine Hospital Service as superintendent of a hospital at Solomon's Island, also as a volunteer cooperative observer for the Baltimore office of the United States Weather Bureau for over thirty-eight years. He was a life member of the Association of Military Surgeons of the United States.

In 1939 Dr. Marsh retired from practice following a fall in which he broke his hip. He was one of the oldest alumni of the School of Medicine of the University of Maryland.

Alexander, Thomas Branch, Scituate, Mass.; B.M.C., class of 1901; aged 65; died, August 28, 1941, of carcinoma of the prostate.

Ashworth, Benjamin Lee, Marion, N. C.; P. & S., class of 1892; aged 74; died, October 19, 1941, of myocarditis.

Avidan, Maurice Simon, Newark, N. J.; P. & S., class of 1910; served during the World War; aged 53; died, October 17, 1941.

Avis, Woodburne R., New Haven, Conn.; P. & S., class of 1894; aged 75; died, November 16, 1941, after a lingering illness.

Berryman, Rozell, Baltimore, Md.; B.M.C., class of 1893; aged 80; died, October 1, 1941, of carcinoma of the stomach.

Brown, Joseph, Des Moines, Iowa; B.M.C., class of 1910; aged 55; died, September 5, 1941, at Hollywood, Calif.

Carroll, Isaiah Francis, Springdale, Conn.; B.M.C., class of 1906; aged 67; died, August 28, 1941, of gastric hemorrhage, cirrhosis of the liver, and pernicious anemia.

Cole, Irving Delbert, Clarksburg, W. Va.; P. & S., class of 1908; aged 60; died, August 10, 1941.

Eddy, Alexander Lough, Greensboro, Pa.; P. & S., class of 1902; aged 69; died, August 20, 1941, in New York.

Ellingwood, William A., Rockland, Maine; class of 1908; aged 58; died, August 30, 1941.

Getz, Charles, Baltimore, Md.; class of 1879; aged 85; died, September 6, 1941, of pneumonia.

Hamaker, William Bernhart, Lancaster, Pa.; B.M.C., class of 1905; aged 63; died, July 15, 1941, of cerebral hemorrhage.

Hanrahan, James M., Elizabeth, N. J.; P. & S., class of 1910; aged 57; died, September 1, 1941, of cerebral hemorrhage.

Harrington, Francis J., Fall River, Mass.; class of 1915; aged 50; died October 14, 1941.

Heitsch, Hubert Messner, Washington, D. C.; class of 1922; aged 47; died, September 17, 1941, in Hartford, Conn., of coronary occlusion.

Hiatt, Houston Boyd, Oldsmar, Fla.; class of 1907; aged 54; died, August 28, 1941.

Hodges, Joseph M., Boone, N. C.; B.M.C., class of 1904; aged 75; died, July 18, 1941, of myocarditis.

Huey, John Fred, Birmingham, Ala.; P. & S., class of 1887; aged 75; died, August 14, 1941.

Johnston, John Kent, Tallahassee, Fla.; class of 1912; served during the World War; aged 53; died, July 15, 1941, of heart disease.

Keylor, Josiah B., Cochranville, Pa.; P. & S., class of 1885; aged 83; died, October 17, 1941.

McGehee, John Webster, Reidsville, N. C.; class of 1904; aged 61; died, September 23, 1941, of coronary occlusion.

Merritt, James Black, III, Easton, Md.; B.M.C., class of 1907; aged 58; died, September 3, 1941, of coronary thrombosis.

Morrissey, William Thomas, New Britain, Conn.; P. & S., class of 1909; served during the World War; aged 59; died, October 23, 1941, of coronary thrombosis.

Newbarr, Bernard Bruce, Los Angeles, Calif.; B.M.C., class of 1911; aged 55; died, July 4, 1941.

Nichols, Frederick Norman, Denton, Md.; class of 1902; aged 62; died, August 22, 1941, of multiple myeloma.

Olsen, Raphael Smith, Lakeside, Ore.; P. & S., class of 1913; aged 56; died, August 11, 1941.

Patton, James Murray, Vandergrift, Pa.; P. & S., class of 1885; aged 83; died, August 5, 1941.

Pole, Edgar A., Hot Springs, Va.; B.M.C., class of 1897; aged 71; died, July 19, 1941.

Sellers, Harry Hallowell, Minneapolis, Minn.; B.M.C., class of 1893; served during the World War; aged 71; died, August 15, 1941.

Shackelford, Jesse M., Martinsville, Va.; B.M.C., class of 1891; aged 71; died, October 2, 1941, of cerebral hemorrhage.

Shaw, Guy Allen, Loreauville, La.; P. & S., class of 1890; aged 72; died, July 23, 1941, of coronary sclerosis.

Sledge, George R., Parksley, Va.; class of 1903; aged 61; died, October 3, 1941, of coronary thrombosis.

Smith, Manly Coke, Simpsonville, S. C.; class of 1913; served during the World War; aged 53; died, August 21, 1941, of cerebral hemorrhage.

Smoot, James Henry, Woodstock, Va.; class of 1892; aged 74; died, September 25, 1941, of tuberculosis.

Wiener, Joseph, Asbury Park, N. J.; class of 1925; aged 39; died, September 8, 1941, of coronary thrombosis.

Zimmerman, George Abram, Harrisburg, Pa.; B.M.C., class of 1910; served during the World War; aged 61; died, August 15, 1941, of cerebral hemorrhage.

BULLETIN
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UNIVERSITY OF MARYLAND

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VIRUSES, VIRUS PROTEINS AND THEIR SIGNIFICANCE*†

JAMES G. McALPINE, Ph.D.
BALTIMORE, MD.

INTRODUCTION

Although a real knowledge of viruses did not begin until the work of Beijerinck a little over forty years ago (4), marked advances have been made in this field, especially within the past two decades. Zinsser (20) writes, "Students of infectious disease are living at the present time in an era which is comparable in rapidity of discovery and intellectual adventure to that which transformed medicine in the period between Pasteur and Ehrlich," and he ascribes this for the main part to the strides which are being made in the study of virus diseases.

For more than twenty years after Beijerinck's work little could be accomplished because the necessary technic and laboratory apparatus had not been developed. A change began in the decade starting in 1920, when newer methods and implements became available. Now the field of endeavor has attained such size that it belongs to the specialist and not within the domain of general bacteriology.

The data which have been collected on viruses from the epidemiologic, the immunologic, the biochemical and physical points of view have changed radically certain fundamental biologic ideas. Perhaps none has had a severer impact than the experiments conducted by Stanley (14), who demonstrated that some filterable viruses were crystallizable proteins and not exceedingly small micro-organisms. These findings have had a tendency to modify the dictum of Koch and his followers that infectious diseases are caused only by living organisms or the poisons which they elaborate. In

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* From the Department of Bacteriology, School of Medicine, University of Maryland.
† Received for publication March 2, 1942.

other words, it was a radical change of the theory upon which bacteriology and public health procedures relating to the control of infectious diseases have been founded, i.e., biogenesis or no life except from previous existing life. If crystallizable proteins are endowed with attributes which have been considered as being present only in living matter, then the conceptions of both the nature and origin of life must be changed.

SOME PHYSICAL AND CHEMICAL CHARACTERISTICS OF VIRUSES

Rivers (12) pointed out that it is impossible to give a positive definition of a virus or to state briefly why diseases produced by them are placed in a separate group from that which contains those produced by other infectious micro-organisms. Primarily, this is a result of the fact that as yet the exact nature of viruses is unknown. Data are available on some of their physical and chemical properties, and speculations as to their nature can be made from these. A brief resume of such characteristics is given below.

Size. The minuteness of viruses is their most striking characteristic, and it is from this that they have derived the name "filterable" or "ultramicroscopic," the latter being the preferable term. Pasteur, in his studies on rabies, was the first to suggest that there might be infectious agents that were smaller than bacteria and could not be seen by ordinary microscopic procedures. Iwanowski (7) demonstrated that the juice of a plant infected with the tobacco mosaic was still infectious even after passage through a filter capable of removing all visible bacteria. However, he did not appreciate the significance of his findings. Beijerinck (4) repeated Iwanowski's experiment and proved conclusively by serial passage that the filtrate was infectious and could reproduce under proper conditions, thus definitely excluding in this case the idea of filterable bacterial toxins. He considered the infectious material to be an exceedingly minute living entity. Almost simultaneously Loeffler and Frosch (9) showed that the etiologic factor of foot and mouth disease possessed similar characteristics. Since then a large number of the maladies of man, animals and plants have been shown to be caused by viruses so exceedingly small that they cannot be seen by the highest power of the ordinary microscope, and that they would in most cases pass through filters which would retain the extremely minute bacterial forms.

The older methods for determining the size of ultra-microscopic viruses were unsatisfactory because of their inherent inaccuracies. Clay and diatomaceous earth filters were employed, and attempts were made to compare the filterability of viruses with that of bacteria, protein molecules and colloidal particles. Now it is known that the pore size in such filters does not determine whether or not the particle will pass through. According to Mueller (10), "Surface adsorption, probably not a matter of electric charge, offers perhaps the most fundamental difficulty. In general, such filters are negatively charged as are virus particles at a slightly neutral or slightly alkaline reaction." It has been shown that viruses can be taken out of suspension by shaking with the powdered materials of which these clay or silica filters are made. Furthermore, the fluid in which these viruses is suspended is an important factor in their filterability; filtration takes place less easily in a saline suspension than it does when broth is the suspending fluid.

In 1927, Zinsser and Tang (19) demonstrated the feasibility of permeable colloidal membranes, and Elford (6) and Bauer and Hughes (3) refined the technic. By this method the sizes of a large number of viruses have been determined. According to these measurements, the sizes range from 125 to 175 millimicrons for vaccinia virus to 8 to 12 millimicrons for that of foot and mouth disease.

Centrifugation has been employed as a check on the measurements made by the use of colloidal membranes. Ordinary centrifuges may be used for the larger viruses, but the apparatus devised by Svedberg is the ideal. Owing to the cost and other difficulties the development of a cheaper and more easily operated centrifuge was essential, and this was accomplished by Bauer and Pickles. Stokes' equation is utilized in computing the results and this requires among other factors the true density of the objects whose size is being measured. As Rivers (12) reports, "Information on the density of viruses is meager and that already in existence needs confirmation. To determine the size of particles by means of the centrifuge it is essential to know their density as well as their shape. Consequently, some of the figures already obtained by centrifugation for the size of viruses may have to be changed to comply with new information regarding the density and shape of the active agents."

Shape. Inasmuch as this characteristic is important in determining size, as mentioned previously, it will be discussed briefly. The difficulties in ascertaining the shape of an invisible so-called micro-organism are obvious and manifold. Following the work of Takahashi and Rawlins (17) on the stream double refraction of flow, the fact that viruses might be in the form of rods, ovals or leaves has been utilized. Certain results have been obtained which need confirmation.

The measurement of exceedingly minute particles was attempted when studying the larger viruses by means of mordants, stains, and the ordinary microscope. Nevertheless, such agents enlarged the particles and the results obtained were relatively useless. Dark ground illumination will reveal the presence of elementary bodies. Barnard (2), by employing fluorescence microscopy, has been able to obtain photographs whereby the size of various elementary bodies can be measured. *Electron optics* are now being utilized for this purpose with excellent results.

Resistance. Formerly it was considered that viruses could be set off from bacteria and protozoa by their differential resistance to various physical and chemical agents. Especially was this true of their supposed relative stability to glycerine. Now it appears that in dealing with viruses there is an extremely heterogeneous group with respect to their reactivity towards various chemical and physical conditions.

Cultivation. Pathogenic viruses which have been studied so far will not grow without the presence of living cells. This characteristic they share with the Rickettsias. It is an example of extreme obligate parasitism if they are considered as biologic entities. Because they grow and multiply only within the living cell, a study of their metabolic activities appears almost hopeless. Some data have been accumulated but the sum total is meager. As yet it has been impossible to demonstrate respiration or sugar utilization in any virus.

Immunity. The question of immunity in virus diseases has proved to be a fascinating problem. Since some of the most spectacular of these maladies, for instance smallpox, appear to produce a lasting immunity, it was assumed that the immunologic reactions in this group differed from those induced by other diseases. Furthermore, it was considered impossible to produce artificial resistance to them unless living viruses were used. Recent experiments have caused a revision of the earlier conceptions. Rivers (13) writes, ". . . . the basic principles underlying serological and immunological phenomena in all fields of biology are identical." In virus diseases the long persistence of immunity which is encountered in smallpox and yellow fever is possibly a result of the continued presence of the virus within certain cells, thus constituting a latent infection which immunizes without producing overt symptoms. It is an extreme example of obligate parasitism in which an almost symbiotic relationship has been established with the host.

In other virus diseases, such as influenza and the common cold, no long term immunity is developed. Rivers (13) feels that a possible explanation for this is the type of tissue involved. When that tissue is superficial an enduring immunity does not result because the tissue itself is temporary. As he says, "The superficial cells lining the respiratory tract are thrown off at regular intervals to be replaced by new cells from deeper layers and do not provide a permanent abode for viruses."

Relative to the use of vaccines in the prevention of this group of diseases, it had been thought, as already noted, that an active immunity could only be induced by a living but usually attenuated virus. Here, too, a revision in ideas has taken place. For certain virus diseases an immunity can be built up by the use of inactivated agents. However, larger quantities must be used and the immunity induced does not persist over long periods of time, thus necessitating repeated vaccinations.

VIRUS PROTEINS

Stanley (14) was able to obtain a crystalline substance, protein in nature, from plants infected with the tobacco mosaic. For years since Allard (1) had worked with this virus it was realized that it was relatively more stable than the other members of the group. Although crystalline preparations had been obtained from the tobacco mosaic virus by Vinson (18), they were shown to be impure compounds. By modifying the various technics Stanley was able to isolate a pure crystalline protein. This was made possible by repeated precipitations with 0.4 saturated ammonium sulfate and absorption on celite.

Repeated precipitations have shown with subsequent recrystallization that this protein is the virus. It is a large nucleoprotein molecule, and by various methods structural changes may be caused and loss of the virus activity results; this activity may be regained by reversing the structural changes. Stanley (16) by numerous experiments has demonstrated that the virus activity and the crystallized protein are one and the same. He concludes, "These results demonstrated that in every case the nucleoprotein isolated was characteristic of the infecting agent regardless of the host, the virus or strain of the virus." In discussing the inseparability of the virus from the crystallized protein he applies Occam's razor, and maintains that "From the practical standpoint it is obvious that it is absolutely unnecessary to postulate the existence of two entities when one is sufficient to satisfy the data."

Since his original work it has been possible to isolate many other viruses in the crystalline form. Stanley (15) mentions a number of plant viruses and also the Shope's papilloma virus, that of equine encephalitis, a coli and staphylococcus bacteriophage, that of Rous sarcoma, and that of foot and mouth disease. This does not constitute the complete list because others have been and are being isolated.

Some viruses are more susceptible than others to chemical manipulation and do not lend themselves readily to the methods employed by Stanley. The vaccinia virus has been separated in a relatively pure state by differential centrifugation. Beginning with the work of Ledingham (8), this has been amplified by that of Craigie (5) and Parker and Rivers (11). Chemical analyses and studies on metabolic activities have been instituted. It would appear that the vaccinia virus has been highly purified.

GENERAL DISCUSSION

From the study of the characteristics of viruses in general and those of various ones in particular it would appear that they do not constitute a homogeneous group. They range in size from the exceedingly minute virus of foot and mouth disease to those approximating the smaller forms of bacteria. In their resistance to chemical and physical agents they exhibit wide differences. The immunity induced by them varies from a solid and, in some cases, a lifelong condition, to one of short duration. Even with these wide differences it is impossible to place them in subgroups because as yet information on the subject is too meager.

Also, as yet sufficient knowledge to define their nature has not been acquired. Here there are three points of view. First, is the one that they are ultramicroscopic living entities distinguished only by size from the bacteria. This was the idea of the older workers in the field and when applied to the larger viruses may be the true answer. On the other hand, the work of d'Herelle on the bacteriophage and its spontaneous appearance in cultures caused some individuals to consider, as Mueller (10) expresses it, "The viruses are nonvital enzyme-like materials produced by certain cells under suitable conditions, notably when stimulated by a small inoculum of the agent itself." Stanley (14) manipulated his crystalline protein from the tobacco mosaic infected plants by methods that had been applied to enzymes and added an impetus to this idea. Lastly, there are many who believe that viruses are intermediate entities between free-living bacterial cells and inanimate substances. This group thinks that by a process of more and more obligate parasitism the viruses have lost certain functions of the ordinary cell and must use products elaborated by the host cells for growth and reproduction. Perhaps all of these assumptions may be true and one is applicable to each virus.

With regard to the findings of Stanley and their effect on ideas concerning the nature of life and its origin nobody can assess their full import at present. It is difficult to decide whether or not the crystalline protein is composed of animate or inanimate substance, since, being able to reproduce, it has one of the attributes ascribed to life. Furthermore, this again postulates the possibility of abiogenesis. In reviewing the ideas of Aristotle and Spinoza, Stanley (16) says, "In order to have spontaneous generation there must be a difference between living things and inanimate things, and despite the general acceptance that a difference does exist and the building on the one side of two schools of thought, vitalism and mechanism, a few feeble although penetrating voices have cried out on the other side." Some have believed that all matter possesses different degrees of life according to the complexity of its organization, and this theory may assist in visualizing the place of these virus proteins in nature.

CONCLUSIONS

1. Great strides have been made in the study of viruses during the past two decades.
2. Stanley's discovery of a crystallizable protein possessing the character-

istics of the tobacco mosaic virus has caused a revision in the ideas on the nature and origin of life.

3. The characteristics displayed by the various viruses indicate that they are a heterogeneous group.
4. The three theories on the nature of viruses are discussed.

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TREATMENT OF THE MENOPAUSE*†

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The menopause represents the physiologic termination of fertile life of the female sex organs. Throughout active reproductive life the ovarian hormones and the anterior pituitary gonadotropic factors have been carefully balanced against each other so that a regular cycle was maintained. The ovarian follicular hormones or estrogens appear to be of particular importance in checking excess pituitary activity. At the climacteric in the normal individual it may be assumed that there are compensating measures occurring in the endocrine balance which make up for the decrease in ovarian activity. The ovary itself may continue to function, but to a lesser degree. Other glands of internal secretion, such as the adrenals, may take over the function of inhibiting excess anterior hypophysial action. As long as the balance remains stable, the patient has no symptoms. In some individuals this normal compensation does not take place, the balance is upset and the untoward symptoms of the menopause develop.

For many decades the active principles of the sex glands have been sought fruitlessly. It is only in relatively recent years that physiologic research has provided the means of accurately extracting the ovarian hormones. These hormones have been successful in alleviating the sufferings of the menopause patient with an endocrine imbalance. The commercial preparation of such drugs is costly, their standardization is difficult, and they are effective only when administered hypodermically. On the basis of misinterpretation of the experimental effects of these substances upon animals, misleading statements have often been made as to their effectiveness in the human patient. Because of such factors many physicians have felt that the use of these hormones is not justified. The introduction of a new synthetic estrogenic drug, diethylstilbestrol, has greatly simplified the treatment of the climacteric. Stilbestrol is inexpensive and is effective when administered orally. Since the use of natural estrogens can be replaced almost completely by this drug, it is felt that the following report of the method employed by the author in treating the menopause should be of interest. The patients are handled in the endocrine clinic of the outpatient division of the Department of Gynecology at the School of Medicine, University of Maryland.

Every candidate for the endocrine clinic is first subjected to a thorough

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gynecologic examination. Her complete history is taken, with emphasis on the menstrual and marital portions. Examination is made of the breasts, abdomen, and pelvic organs. A urinalysis is done. Care is taken to exclude the possibility of carcinoma in any organ. The sex hormones are at least potentially carcinogenic and with their use must be considered the hazard of inciting or aggravating a malignant lesion. A search is made for any purely gynecologic disorders that may be present. These, particularly the pelvic relaxations, are not infrequent and are often classed by the patient as part of her change of life. Upon completion of the preliminary examination the patient is transferred to the endocrine department for classification and treatment.

The diagnosis of the menopause is established by a history of progressively decreasing menstrual periods, with eventual cessation. A wide range of time interval and frequency of menses constitute the normal change of life. This must always be on the negative side; any increase in duration of flow or in frequency must be considered pathologic until proved otherwise. Few patients develop true symptoms until they show evidence of the lessening of menstruation. The majority of women at the climacteric exhibit no symptoms. When the symptoms are few or none and when the symptoms will respond to simple measures, sex hormone therapy is not indicated. Reassurance is given. The patient is instructed as to the necessity of periodic examinations and advised to report promptly any symptoms that may be suggestive of carcinoma, particularly any recurrence of bleeding and the development of any sort of leukorrheal discharge. Postmenopausal vaginal bleeding or leukorrhea always must mean carcinoma until another diagnosis is made.

All patients with untoward symptoms of the menopause, regardless of their severity, are placed first upon a preliminary trial of simple medical and hygienic measures. This is continued for at least two weeks before recourse is had to hormone therapy. A daily sedative is prescribed. One-half grain of phenobarbital is given three times daily, but any mild sedative will do. All purely medical and symptomatic complaints are investigated and remedied. If the patient is overweight, a reducing diet is in order. Arthritis is frequent in the climacteric and physiotherapy may be indicated. Arthritic symptoms do not respond to hormone treatment. The presence of numerous functional complaints may indicate a poor prognosis as to the relief of symptoms. If the patient is actually psychotic, therapy is absolutely contraindicated and the case is referred to the psychiatrist. Many patients with really severe symptoms will respond satisfactorily to these simple measures and in such cases no further treatment is necessary. When the complaints are not relieved, even though they may be considered largely functional in origin, at least one adequate course of endocrine therapy is administered.

One subjective symptom alone indicates the severity of the menopausal disturbance and in addition can provide an accurate index of the response to treatment. This symptom is the vasomotor flush so typical of the condition. A typical flush begins about the chest and shoulders and extends upward over the neck and face as an enveloping heat wave, with great rapidity and discomfiture to the patient. If the description of the flush when related by the patient is not similar to this picture, it is believed that the flushes are not true ones and are probably functional in origin. It is especially rare to find them beginning at the head and extending downward to the extremities. Other symptoms, such as vertigo, headaches, palpitation, nervousness and indigestion are of secondary importance and in the absence of hot flushes seldom will respond to therapy. The frequency of flushes during the day and night is recorded. Night flushes are those which awaken the patient from her sleep and are usually indicative of rather severe symptoms. Three arbitrary classifications of the severity of the symptoms of the menopause are made. A mild case exhibits anywhere from an occasional weekly or monthly flush to three or four a day. A moderately severe case develops from four to ten daily, with or without an occasional night flush. A severe case demonstrates more than ten flushes daily, with regular night flushes. Some patients will complain of them every five or ten minutes. This is especially true of patients with an artificial menopause.

The drug of choice to be used in treating the climacteric is oral diethylstilbestrol, commonly called stilbestrol. This is a relatively new synthetic drug which is fully effective when given by mouth. No advantage is to be had by intramuscular injection in an oily solution. The drug is sold by many commercial pharmaceutical houses and is supplied in 0.1, 0.5, 1.0, 5.0 and 10.0 milligram tablets, usually enteric coated. In treating the menopause there is no need for larger daily doses than one milligram.

The initial dose necessary is either one-half or one milligram administered daily. This dose is maintained for about four weeks. Symptomatic response usually occurs within one to three weeks. When the patient has been relieved satisfactorily the initial dose is continued for another seven to fourteen days. The amount is then reduced by one-half and continued for another two to four weeks. A rest interval is then allowed and no further treatment is given until the patient complains of a recurrence of symptoms. She is then given a supply of one-half or one milligram tablets and instructed to reduce her dosage to the smallest weekly or monthly ration that will keep the symptoms under control. In most cases a few tablets a month will be sufficient. Often after several months no medicine at all will be necessary. If a continuous maintenance dose is necessary an attempt is made to reduce it to the smallest or one-tenth milligram tablet to keep the patient comfortable. There is no contraindication to prolonged treatment over a long period of time. Several patients with severe symptoms of artificial menopause have been taking continuous small amounts of this drug for periods of time up to four years without ill effects.

A few patients will develop nausea from intramuscular as well as oral administration of the drug. This represents an idosyncrasy to the drug and is not evidence of toxicity. It is countered by allowing a few days' rest from treatment and then beginning again with one-half of the former dose. If nausea redevelops, the dose is again divided. By doing this tolerance can be established in most patients, even to the extent of increasing the drug to the original amount without a recurrence of nausea. Only a few patients exhibit intractible intolerance. In the future alternate synthetic oral estrogenic drugs for oral administration may be available but these are still in the experimental stage. If the nausea is intractible or if the patient is one of the few who do not respond symptomatically to stilbestrol, recourse should then be had to the use of natural estrogenic products by intramuscular injection.

The patient whose uterus is still intact must be cautioned that bleeding may occur during treatment or after cessation of the drug. This bleeding is of the type produced by the administration and withdrawal of any of the estrogenic hormones, natural or artificial. Daily doses of less than one-half milligram of stilbestrol seldom produce bleeding. This bleeding should not recur when the quantity is reduced to a small weekly or monthly maintenance dose. In any event, it is no cause for alarm and will subside quickly when the drug is stopped. If bleeding continues for more than a week after the drug is discontinued or if bleeding recurs some time after therapy has ceased, a careful pelvic examination and diagnostic curettage are indicated to rule out the presence of an organic lesion.

Natural estrogens are supplied in two forms. One is a solution in oil of the pure crystalline forms of the three primary estrogens or their derivatives. These primary forms are estrone, estradiol and estriol. The other preparation is a solution of mixed estrogens in oil which is not purified to any one primary hormone. Both types are about equally effective in the human patient. Much contradictory work has been done to prove that one form of estrogen is more effective than another. This work has been performed on laboratory animals but has not been reasonably proved to hold true for the human, although each manufacturer can cite voluminous material in favor of his own product. In the author's hands the solution of mixed estrogens has been as effective as any and is usually less expensive than the more highly purified forms. The unit of dose is the international unit. One international unit represents the equivalent in biologic activity of one gamma or microgram of pure crystalline estrone. Speaking figuratively, this may be represented as approximately the minimum dose necessary to produce estrus changes in the vaginal mucosa of one rat. Any estrogenic preparation and any ovarian extract to be used for estrogenic stimulation which is not standardized in terms of international units should not be used. Even though such preparations may be actively estrogenic there is no index of their activity given by which an accurate dose factor can be established, and time and money may be wasted in employing them. There is no reason whatsoever for the use of nonestrogenic ovarian preparations in treating the menopause. The solutions of estrogenic hormones in oil are provided in ampules in varying strengths from 1000 to 20,000 or more international units per cubic centimeter. The patient is given a biweekly or triweekly dose of 10,000 international units (i.u.) intramuscularly over a period of six weeks. This makes a total dose of from 120,000 to 180,000 i.u. Response usually begins during the second week of treatment and becomes progressive. A cumulative effect will often be noted which will persist for several weeks after treatment is ceased. When the response has been slow, one weekly injection of 5000 to 10,000 i.u. is continued for another two to four weeks. A rest period is then allowed and no further therapy administered until symptoms recur. If this happens, stilbestrol is again administered in small weekly doses, either alone or in combination with sedatives. No further intramuscular injections are given unless they are necessary to relieve recurrent symptoms and then they are made as small in dose and as far apart as the patient will tolerate without complaint. Again it must be emphasized that treatment with natural estrogens is needed in but very few patients, and once these have had a satisfactory course of intramuscular injections further symptoms are usually controllable by small doses of oral stilbestrol or by sedatives.

There are numerous preparations of the natural estrogens marketed in tablet and liquid form for oral use. These require enormous doses to be at all effective by mouth for they are destroyed by the liver after being absorbed. Such therapy is expensive and in the author's experience *is not justified.*

Occasionally, after completion of a course of therapy with natural estrogens withdrawal bleeding will be noticed. This is much less likely to occur with the use of these preparations than it is with stilbestrol. Even rarer is the occurrence of bleeding during a course of treatment. When such bleeding occurs it is managed exactly as has been described previously under the use of stilbestrol.

A few patients complain of breast discomfort while they are receiving estrogenic therapy. This also will be noted more frequently when the patient is receiving the synthetic estrogen than when the natural form is administered. It rarely becomes severe enough to inconvenience the patient and will disappear a few days after treatment is discontinued.

Reference should be made to the subcutaneous implantation of pellets of stilbestrol and the estrogens through a trocar or a small incision. This form of therapy has been extremely successful in the hands of experienced investigators, but is still in the experimental stage. The methods have not been standardized sufficiently and the technical details are too complex for the average practitioner. It has no advantages to offer over orally administered stilbestrol. Nothing is easier than swallowing a pill once a day. Oral administration can be discontinued at any time, whereas it requires a minor operation to remove the pellet.

Some investigators have employed male sex hormones or androgens in the treatment of the menopause, particularly in those patients not responding well to estrogenic therapy. In time this may have a rational place in treatment of the climacteric; at present it is in the experimental stage. Until the changes produced by these hormones in the female patient are better understood the androgens should not be employed except under investigative conditions. With their use is the ever-present possibility of masculinization of the patient. It is not comforting to have a patient grow a beard and develop voice changes while under treatment.

The other sex hormones, the pituitary and chorionic gonadotropines and the corpus luteum hormone, progesterone, apparently have no place in treating the menopause.

A small percentage of patients suffering from the menopause have an associated senile vaginitis. The average patient exhibits moderate atrophy and thinning of the vaginal mucosa but these seldom are sufficient to produce symptoms and consequently need no special treatment. In a few the changes are pronounced, with advanced atrophy which results in the formation of innumerable minute ulcers. These bleed and become infected, so that a discharge occurs. This is the commonest cause of spotty bleeding in the climacteric patient.

Oral and parenteral estrogenic treatment usually produce some improvement in the objective findings of the vaginal mucosa but it is not enough to satisfy the patient subjectively. It is expeditious to supplement general treatment with the use of a local suppository or insert containing 1000 i.u. of natural estrogens. This should be used nightly for two to three weeks and will be found to produce prompt and often lasting relief. The natural estrogens appear to be more effective in their action upon the senile vaginal mucosa than do similar preparations of stilbestrol applied locally.

SUMMARY

Treatment of the menopause when conducted in a systematic manner is highly satisfactory. Oral stilbestrol is indicated as the drug of choice. In a few patients the intramuscular injection of natural estrogens may be indicated. Subjective hot flushes are the only reliable index of the severity of symptoms. Senile vaginitis as an accompanying condition to the climacteric is best treated with local suppositories of natural estrogens.

PEPTIC ULCERATION OF THE CARDIA OF THE STOMACH: A ROENTGENOLOGIC CONSIDERATION* †

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In recent years the roentgenologic exploration of the cardia of the stomach has become a matter of keen interest to the roentgenologist. Very little attention has been given to the roentgen technic as one of the important factors in the failure to demonstrate peptic ulceration in the cardia of the stomach. Roentgenologic difficulties in the diagnosis of peptic ulcer of the cardia of the stomach arise often enough to reveal the inadequacy of some of the roentgen methods of examination. Notwithstanding these difficulties, however, a great deal of uncertainty regarding the normality of the cardia of the stomach may be dispersed by a painstaking roentgen study. Since interest is centered chiefly in the demonstration of the anatomic changes produced by the ulcer, the present methods of examination of the cardia have been greatly augmented by the spot film technic. This presentation is primarily concerned with a discussion of the roentgenologic diagnosis of peptic ulcer situated in the cardia of the stomach, as well as hemorrhage produced by such ulcers.

By far the greatest majority of gastric ulcers occur on the lesser curvature or on the walls close to the lesser curvature of the body of the stomach. Owing to their position, these ulcers are easily demonstrated by means of the roentgenographic examination. However, ulcers in the cardia of the stomach are not so easily depicted and, unless they are especially sought for, may elude recognition. This is especially true of those ulcers which are located on the walls of the cardia.

Although the incidence of gastric ulcer situated in the cardia of the stomach is low, the roentgenologic incidence of demonstrating the ulcer crater falls into still a lower scale. Ulcers of the cardia of the stomach are more often overlooked than those which occur in any other portion of the stomach. Even though a painstaking roentgenographic examination is made, the ulcer niche defect may not at all times be demonstrable. It is true that peptic ulcer of the cardia of the stomach is diagnosed more frequently now than in the past, but there is still a small percentage of cases in which the condition is entirely overlooked. Peptic ulcer of the cardia of the stomach occurs more frequently on the walls of the stomach and often close to the eso-

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phageal orifice. These ulcers are best observed in oblique or lateral views and may not be visible in the roentgenograph or fluoroscopic examination made in the prone or supine position. Failure to obtain a view of the stomach in all angles, especially under the fluoroscope, explains why in many instances ulcers of the cardia are not recognized. Special attention must be directed to the use of the erect posture in obtaining information with regard to these lesions situated in the cardia. The filling defect and other roentgenologic criteria of ulcerating lesions frequently are best demonstrated in the erect posture. In this position the cardia of the stomach is shown in relief and the mucosal picture is more clearly seen than when the examination is made in the recumbent posture. In the roentgenographic examination all of the appropiate positions, including the erect spot film technic, should be employed in order to obtain the necessary information for the diagnosis of a cardial ulcer. At times the examination should also be made in the Trendelenburg position in order to completely fill the cardia of the stomach. It is important to observe the first mouthful of the barium meal as it enters the cardia of the stomach, and to note carefully the cardioesophageal and cardial areas. A mucosal study and a spot film made in the erect posture are frequently necessary in order to disclose the ulcer niche defect on the walls of the stomach. Emphasis must be placed on the

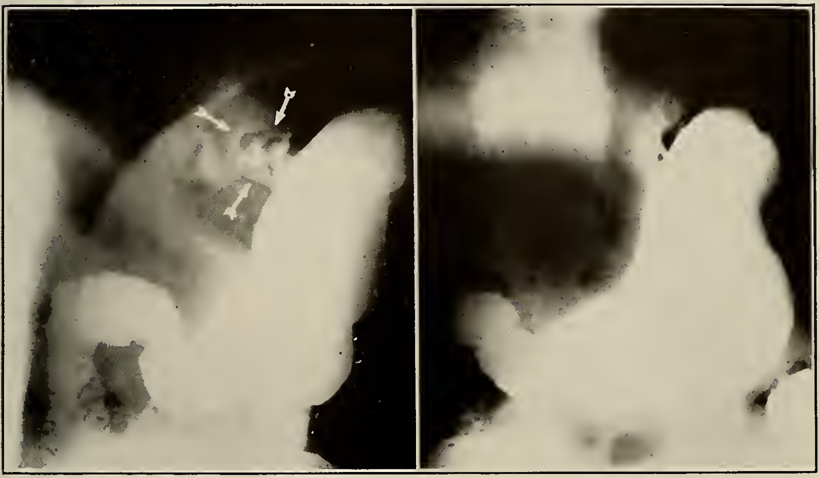
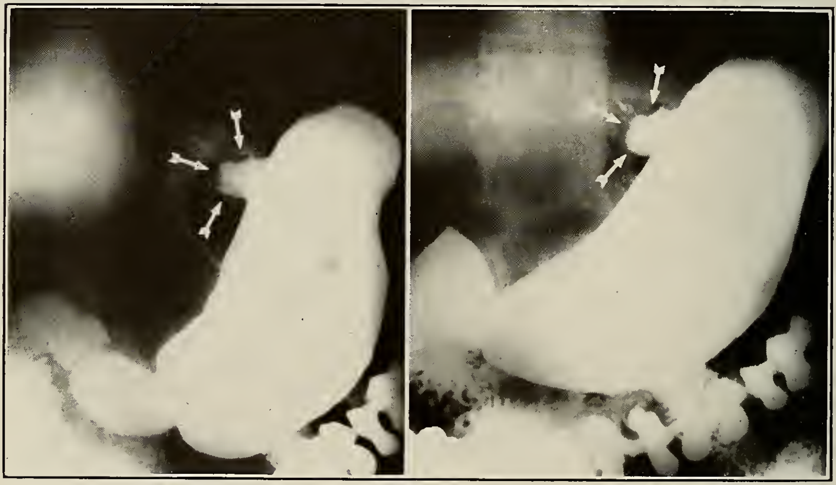

b. a.

FIG. 1. Roentgenograph showing a large gastric ulceration in the cardia of the stomach. *a*, A roentgenograph made in the postero-anterior (prone) position does not reveal the ulcer niche defect. *b*, The same case with the roentgenograph made in the right oblique position shows a large irregularly filled, mottled shadow projecting into the gas bubble, at the arrows, and represents a large ulcer niche defect.

importance of this roentgenographic technic in the examination of the cardia, which should be a routine procedure in all gastric studies.

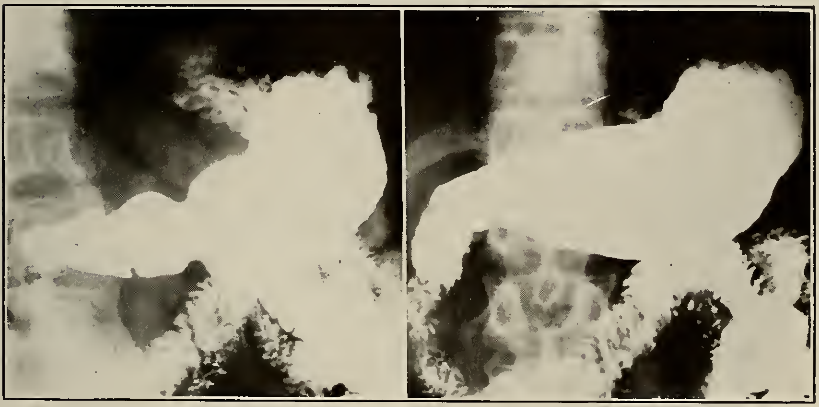
The only anatomic pathognomonic sign of peptic ulcer is the demonstration of an ulcer niche defect. Although there is often a fixed appearance of the curvature or an irregular contour of the air bubble and changes in the mucosal pattern, the diagnosis of a benign peptic ulceration cannot be made with any degree of certainty unless the niche defect is visualized. The reasons why the cardia of the stomach presents many difficulties in the roentgenographic examination are obvious. One of the principal reasons is the fact that the cardia is difficult to manipulate owing to its location behind the costal arch. Because of this the cardiac portion of the stomach cannot be explored fully by the compression technic. Another important cause for failure to recognize the cardial ulcer is the fact that the roentgenologist often fails to observe the cardial walls during the first intake of barium as it passes from the esophagus into the stomach.



b. *a.*

FIG. 2. The same case with roentgenographs made at a later examination demonstrates the characteristic large ulcer crater niche defect in the cardia of the stomach, in both the postero-anterior (prone), *a*, and the right oblique positions, *b*, at the arrows.

Ulcerations located in the cardia of the stomach vary in size but are often unusually large. It might be worth while to mention the fact that, in the author's experience, unusually large-sized benign ulcers are observed more frequently in the cardia of the stomach than in any other region. These large-sized ulcers are often of the penetrating or chronic perforating type, and are usually surrounded by a large area of induration and edema, with thickened mucosal folds. It is noteworthy to point out that even though these ulcers are large, they infrequently are overlooked. Attention must likewise be directed to the small fleeting type of ulcer niche defect which is sometimes seen in the cardia, especially when it is situated near the cardioesophageal orifice. This type of ulcer is best seen during the examination following the first swallow of the barium meal. The ulcer is visible as a small niche defect or, when greater in size, as a flattened large defect which retains the opaque meal for only a short period of time, while the barium flows over the affected area. The changes may be observed repeatedly with each swallow of barium. On the other hand, when the stomach is completely filled the defect may not be seen. Another interesting point to mention is the demonstration of the ulcer niche in only one position; even a slight rotation of the stomach may eliminate its appearance from the roentgenograph. This accounts for the visualization of the ulcer niche defect in only one or two films in a series of roentgenographs of the stomach. Under the fluoroscope the ulcer niche defect is more persistently seen because the examiner can better control the position of the patient with the position of the ulcer.



b. *a.*

Fig. 3. *a*, Postero-anterior (prone) and *b*, right oblique roentgenograph of a case of gastric ulcer of the cardia of the stomach, showing no roentgenographic signs of ulceration.

The differential diagnosis between the large-sized benign and malignant ulceration in the cardia of the stomach is frequently difficult, and in many instances no definite opinion should be given regarding this aspect. Whereas it is claimed, and justly so, that a gastric ulcer, the size of which measures over 2.5 centimeters in diameter, is more likely to be malignant, this does not especially hold true in ulcers situated in the cardia of the stomach. A large percentage of these ulcers are benign, in spite of the fact that they are often of tremendoùs size. The chief signs of malignancy of the cardia of the stomach, besides the filling defect, are shortening of the curvatures, fixation of the curvatures, changes in contour of the air bubble, rapid emptying of the stomach, and reduction in the size of the stomach. These roentgenographic signs are not observed in the large-sized benign ulceration of the cardia.

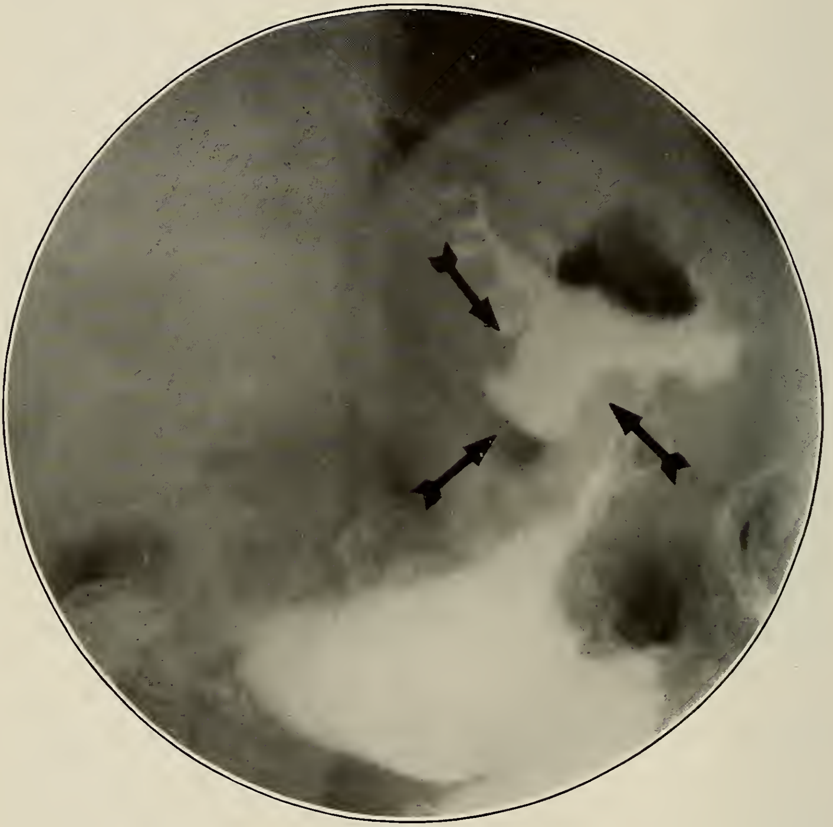


Fig. 4. A roentgenograph made in the erect posture by the spot film compression technic illustrates a large ulcer crater in the cardia, at the arrows.

Ulcers of the cardia of the stomach are a frequent causative factor of gastric hemorrhage. The cardial ulcer, because of the difficulty encountered in demonstrating the niche, is one of the types of bleeding ulcers which are often not recognized following a recent hemorrhage. The incidence of the roentgenologic demonstration and recognition of an ulcer niche in gastric hemorrhage is particularly lower than in the nonhemorrhagic cases. There can be no doubt that many of the hemorrhages are caused by the large ulceration which occurs in the cardia of the stomach. In the author's experience it has been noted that the larger cardial ulcers have a greater tendency to produce gastric hemorrhage than those which are situated in the body of the stomach. On the other hand, it must be emphasized that even small ulcers may cause profuse hemorrhages.

SUMMARY

The roentgenologic diagnosis of gastric ulcer situated in the cardia of the stomach offers many difficulties which are discussed in this presentation. Emphasis is made on the frequency of the larger type of ulcers found in the cardiac portion of the stomach and their tendency to produce hemorrhage. The roentgenologic technic for the demonstration of the cardial ulcer, stressing the importance of observing the first mouthful of barium as it enters the stomach, the need for the erect spot film technic, the value of the fluoroscopic examination, and the frequent failure to demonstrate the cardial ulcer, even though it is of large size, are discussed.

ACUTE PANCREATITIS WITH DIABETIC ACIDOSIS OCCURRING IN THE COURSE OF DIABETES MELLITUS*†

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It is well known that changes in carbohydrate metabolism frequently occur in cases of acute pancreatitis. Although Fitz (1) in his classical paper on acute pancreatitis failed to mention the occurrence of glycosuria, subsequent contributors noted its frequency. Egdahl (2) reported an incidence of 5 per cent in 105 cases. Guleke, cited by Walzel (3), found glycosuria in about 10 per cent of his cases. Shumacker (4) in a comprehensive study of the literature found that glycosuria was reported seventy-eight times in 700 cases, an incidence of 11 per cent. Hyperglycemia is another manifestation of altered carbohydrate metabolism in acute pancreatitis (5, 6). Shumacker found that twenty-eight of fifty-five cases had blood sugar levels above 200 mg. per 100 cc. It is also known that a small percentage of the patients with acute pancreatitis become permanently afflicted with diabetes (7).

The cause of these disturbances in carbohydrate metabolism is not well understood. Brocq and Verangot (6) thought that the large amounts of trypsin liberated in acute pancreatitis might have some inhibitory action on insulin. Gray and his co-workers (8) expressed the opinion that the variation from normal levels may be the result of some degree of hepatic dysfunction. In spite of the fact that three-quarters of the human pancreas may be removed without demonstrable changes in carbohydrate metabolism (9), it is still assumed commonly that the inflammatory process destroys or causes sufficient dysfunction of islet tissue to precipitate a deficiency of insulin.

Warren (10) in a study of the pancreas in 484 cases of diabetes mellitus found acute pancreatitis in four cases, pancreatic apoplexy in two, and subacute pancreatitis in six instances. In 121 cases of death during coma in his series, acute or subacute pancreatitis was associated with eleven cases, an incidence of over 9 per cent. Gibb and Logan (11) in 147 autopsies on diabetic patients found acute inflammatory changes of the interstitial pancreatic tissue in six cases, an incidence of 4 per cent as quoted by Root. Hemper (12) found sixty-seven cases of acute necrosis of the pancreas in 20,000 autopsies, or 0.3 per cent.

This increased incidence of acute pancreatitis in diabetic patients raises the question as to whether diabetes mellitus may not in some way be a pre-

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disposing factor in the production of this disease. Rich and Duff (13) in a series of nineteen cases of acute pancreatitis found that two of the patients had previously been known to have diabetes. These authors referred to the work of Babkin (14), who in 1935 reported that hyperglycemia in rabbits results in the production of an extremely ferment-rich pancreatic juice. If the theory of obstructed pancreatic ducts as an etiologic factor in acute pancreatitis is tenable, then the combination of the obstructed duct with ferment-rich pancreatic juice would warrant the assumption that persistent hyperglycemia is of great importance in determining the course and extent of acute pancreatitis.

Root (12) in 1937 called attention to the clinical importance of acute pancreatitis occurring in the course of diabetes. In autopsies on twenty-six patients who died in diabetic coma he found four instances of acute pancreatitis. This author stressed the grave outlook for patients in whom this combination of processes occurred and said that the clinical diagnosis of acute pancreatitis in diabetic coma was rare. He also noted that these patients showed marked degrees of fatty infiltration of the liver and that the pathologic process in the pancreas was of a septic rather than of a hemorrhagic nature.

Foord and Bowen (15) have reported two similar cases with acute interstitial pancreatitis. Schott (16) and Shumacker (4) have each reported a similar case, and Cabot (17), Case No. 16,282, is another example of this process. Beck (18) has reported a case of subacute pancreatitis in a patient previously known to have diabetes who was admitted in coma.

CASE REPORTS

In seventeen autopsies on patients who died in diabetic coma at the University Hospital there have been found three cases of acute interstitial pancreatitis.

Case No. 1: J. S., a white male, age 30 years, was admitted to the University Hospital on December 5, 1939. At the time of his first admission to the hospital in 1933 the diagnosis of diabetes mellitus was established and he was instructed as to diet and the use of insulin. His course since that time was not well known, but he did not adhere to instructions and frequently engaged in alcoholic debauches. It was known that he had received no insulin for the three days prior to admission, and on the day preceding admission could not be aroused. It was felt that there was severe acidosis for at least three days and that he had been in coma for twelve hours.

On admission he did not respond to any form of stimulation. There was evidence of marked dehydration, but he was well-developed and heavily built. The pupils were dilated widely; the eyeballs were soft. Respirations were irregular, jerky and shallow but not typically of the Kussmaul variety. The pulse rate and blood pressure could not be obtained. The heart sounds were feeble at a rate of about 20 per minute. There were a few fine rales over the lower lobe of the left lung. The abdomen was rigid throughout and the bladder was greatly distended. When admitted the blood sugar level was 667 mg. per 100 cc., the carbon dioxide combining power 12 volumes %.

A total dose of 180 units of insulin with parenteral fluids, intravenous soda bicarbonate and Ringer lactate were of no avail and he died twelve hours after admission without regaining consciousness. At the time of death, the blood sugar level was 200 mg. %, and the carbon dioxide combining power 22 volumes %.

At autopsy there was little gross change in any of the organs except the liver, which was pale in color and weighed 2800 grams. The gallbladder was thin-walled and showed no evidence of stone formation or inflammation. The biliary ducts were patent. The pancreas was firm and had a slightly yellowish color. It weighed 250 grams. On its cut surface there was a normal appearance, except for a few pinpoint-sized areas of brownish discoloration. There was no evidence of fat necrosis in the surrounding tissues. Microscopically the liver showed extensive vacuolization of the liver cells, not limited to any part of the lobules. Appropriate stains showed these to be fat vacuoles. Sections of the pancreas showed some increase in interstitial tissue, with considerable areas of fat necrosis, and infiltration with polymorphonuclear cells was also seen in the acinic structures. The islet tissue was fairly well preserved but the islets seemed reduced in number. Some of the pancreatic ducts contained an exudate which was filled with polymorphonuclear neutrophils.

Case No. 2: F. H. was a 52 year old obese colored female, who for one month had noticed polyuria, polyphagia and polydipsia, with weakness and a loss of thirty pounds of weight. She was admitted to the University Hospital on October 3, 1940. For three days before admission she had been drowsy, and for twenty-four hours had been comatose. Her urine had not been examined for seven years and there was no history of abdominal pain, nausea or vomiting.

An examination revealed a deeply comatose female with typical Kussmaul breathing. Her temperature was 100F., pulse rate 120 per minute, and the rate of respirations was 4 per minute. Corneal reflexes were absent; the skin and mucous membranes were extremely dry. The heart and lungs were normal; the systolic blood pressure was 112 mm. and the diastolic pressure 62 mm. There was slight rigidity in the upper abdomen but no masses were palpable. The deep tendon reflexes were not obtained.

On admission the blood sugar level was 1142 mg. per 100 cc., the carbon dioxide combining power 21 volumes %, and the nonprotein nitrogen 109 mg. %. The leukocyte count was 10,400, with 96% polymorphonuclears. The patient was given a total dosage of 1020 units of insulin and over twelve liters of parenteral fluid during the thirty-six hours of hospitalization. She failed to void and on catheterization only 200 cc. of dark urine was obtained. Gastric lavage was performed on three occasions and was always productive of turbid dark brown fluid. The upper abdomen was rigid during the last twelve hours of life, and when the patient died without regaining consciousness the blood sugar level had fallen to 190 mg. per 100 cc. and the carbon dioxide combining power had risen to 46 volumes %.

An autopsy was performed on the body of an extremely obese middle-aged colored female. On incising the abdomen the peritoneal surfaces presented a normal appearance. However, in the region of the epiploic foramen and the transverse mesocolon the tissues were studded with many small areas of fat necrosis. The lesser peritoneal cavity contained about 20 cc. of cloudy fluid; there were several small hemorrhages in the tissue around the pancreas.

The lungs showed evidence of pulmonary congestion and edema and the alveolar spaces were filled with fluid. There were moderate sclerotic changes in the coronary vessels but the heart was otherwise normal. The aorta showed mild atherosclerosis without ulceration.

The liver weighed 1750 grams and was of a yellowish-brown color. A histologic examination revealed considerable fatty changes in the liver cells, especially around the central veins, and a moderate amount of cloudy swelling. There was one localized encapsulated area of necrosis that was not associated with the fat necrosis. The gallbladder was of normal size and shape and contained thick viscid bile, with no stones. The biliary passages were normal throughout.

The pancreas weighed 150 grams and was studded with areas of fat necrosis. It was flabby and slightly hemorrhagic, with ill defined lobulations. Histologically there was considerable interstitial fibrosis with round cell infiltration. There were many areas of fat necrosis with inflammatory changes characterized by numerous polymorphonuclear cells, macrophages and round cells. The acini were distorted but not destroyed. The islands of Langerhans were well preserved and normal in number. The pancreatic ducts were normal. All other organs were essentially normal.

Case No. 3: N. V., a 28 year old white male, was admitted on December 2, 1940. He had diabetes for two years but it was fairly well controlled with diet and protamine zinc insulin. However, for the past year he had not adhered strictly to his dietary instructions. One week before admission he noticed marked lassitude and general malaise. For three days there had been epigastric fulness, anorexia, and frequent belching that was not relieved by sodium bicarbonate. At 10 a.m. on the morning of admission he was seized suddenly with severe cramplike, generalized abdominal pain, most intense just above the umbilicus. This was associated with nausea and vomiting. No shock was present, but the pain gradually increased in intensity and for four hours preceding admission he had been drowsy.

An examination revealed a well-nourished drowsy male with typical Kussmaul breathing, who complained of severe abdominal pain. His temperature was 99F. and the pulse rate 118 per minute; there were 26 respirations per minute. The systolic blood pressure was 108 mm. and the diastolic pressure was 72 mm. There was evidence of considerable dehydration, and the eyeballs were quite soft. The chest was clear; the respirations were abdominal in type. An examination of the abdomen showed moderate tenderness over an area from the umbilicus to the xyphoid process and laterally for 5 cm. in each direction. There was a moderate amount of muscular rigidity.

The blood chemical examination on admission revealed: sugar 339 mg. per 100 cc.; carbon dioxide combining power 16 volumes %; 10,200 leukocytes; cholesterol 666 mg. per 100 cc. The blood diastase determination at the time of admission was 969 units (Somogyi). During his thirty-six hours in the hospital the patient received a total of 1175 units of insulin and 11,000 cc. of parenteral fluid. The carbon dioxide combining power never rose above 20 volumes %, although the blood sugar level fell to 185 mg. per 100 cc. Repeated gastric lavages were productive of a turbid dark brown fluid.

A blood diastase determination twenty hours after admission showed 1528 units, at twenty-four hours, 1198 units, and at thirty-six hours, 1214 units. Respiration ceased after thirty-six hours.

The necropsy was limited to the abdomen through a small incision and was performed on the body of a well-developed white male. There was 200 cc. of cloudy watery fluid in the abdomen.

The liver and gallbladder weighed 2200 grams. The gallbladder was moderately distended by dark green tenacious bile and the biliary passages were entirely normal. The liver tissue seemed darker than normal and the liver lobules were moderately indistinct. The liver cells showed cloudy swelling and were distended with lipoid.

The pancreas was firm and on sectioning showed evidence of edema. Fat necrosis was seen in the interlobar fat and the larger ducts contained a gray purulent material which could be traced to the ampulla of Vater. All sections showed marked infiltration of the interstitial tissues with neutrophils and in some of these areas there was evidence of the beginning of necrosis. The ducts contained necrotic material, neutrophils and desquamated epithelium. There was some fat necrosis in the interstitial and peripancreatic tissue. A majority of the islets showed lesions varying from edema with capillary engorgment to complete replacement of the islets. The kidneys showed evidence of moderate cloudy swelling.

COMMENT

In the three cases herein recorded the blood sugar levels responded favorably to insulin therapy. The carbon dioxide combining power rose in one case, but in the other two it did not respond. Joslin (19) reports that acute pancreatitis should be considered in any instance of diabetic acidosis that does not react favorably to intensive insulin therapy.

In all of these cases the pathologic process in the pancreas was of an inflammatory nature, rather than a necrotic or hemorrhagic type. This is the variety which some authors term the septic or acute interstitial type and is the process which Root has also recorded as being present in his cases. In none of the cases herewith reported was there any disease of the bile ducts or other disease which could have been considered an etiologic factor for the pancreatitis.

The cause of abdominal pain in diabetic acidosis has not been established. The possibility that it may be caused by an early acute pancreatitis has been forwarded by several authors (7, 20). Nevertheless, the frequency with which instances of diabetic acidosis with severe abdominal pain are found at autopsy to have no evidence of acute pancreatitis would seem to refute this premise. In addition, cases of acute pancreatitis have been reported in which there was no mention of abdominal pain (12, 15). However, in one of the reported cases the diagnosis of acute pancreatitis was made because of the persistence of severe abdominal pain and boardlike rigidity.

In the third case, serum diastase determinations were obtained from the time of admission until death and were found to be elevated persistently. The opportunity of studying serum diastase levels in three other cases of diabetic acidosis presented itself. One of these patients complained of severe abdominal pain simulating a coronary thrombosis prior to admission. A blood diastase value of 758 units (Somogyi) was obtained, but at postportem examination no evidence of pancreatitis was found. In another patient normal values for serum diastase were obtained despite the presence of severe pain in the upper portion of the abdomen. A third case of severe acidosis with moderately severe pain in the same section showed a serum diastase determination of 467 units on admission. This rose to 568 units in nine hours, but as the symptoms improved there was a gradual fall until two weeks later a value of 22 units was reported.

A recent extensive study by Heifetz, Probstein and Gray (21) emphasized the importance of determining urine and blood diastase levels. These authors have found that cases of renal insufficiency, which frequently are associated with high blood diastase findings, may be excluded by this method. Since renal failure is not uncommon during diabetic coma this procedure should be a part of the diagnostic study in these cases. Evidences of nitrogen retention should make one cautious in interpreting a single blood diastase determination as evidence of pancreatic disease.

There is little information available in the literature on serum diastase determinations during the course of diabetic coma. However, Lewison (22) has found no elevation of serum diastase in hyperglycemia but rather an inverse ratio between blood sugar and diastase levels. Norby (23) has also reported no increase in blood diastase determinations in diabetic patients. Gray and his co-workers in sixteen cases of diabetic coma found the diastase level to remain below 60, which is the lower limit of normal.

SUMMARY

Two cases of acute pancreatitis with diabetic acidosis occurring in the course of diabetes mellitus are presented. In addition, a third case which probably also falls in this category is reported. In one of these patients the diagnosis was established before death by means of repeated serum diastase determinations.

CONCLUSIONS

1. Acute pancreatitis frequently causes disturbances in carbohydrate metabolism.
2. Acute pancreatitis occurs with greater frequency in diabetic acidosis than is generally realized.
3. Serum diastase determinations may be of value in the recognition of these cases.

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CONGENITAL CYST OF THE URETHRA IN A THREE WEEKS OLD MALE INFANT CAUSING PYONEPHROSIS AND DEATH*†

A Case Report

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Congenital cysts of the urethra, while infrequent and seldom mentioned in textbooks, are not unknown. The majority of these seem to arise from one of Cowper's glands. There are many references in the literature to such cases. They are known to occur in both infants and adults.

Most of the cysts recorded apparently were found at routine autopsy, especially by European investigators such as Elbogen (1), Englisch (2), Gubler (3), Fenwick (4), and others, in the latter part of the nineteenth century.

Within the past few years other cysts of congenital origin have been reported and discussed by such American writers as Campbell (5), Wesson (6), and Johnson (7). Some of these workers have found cysts in the embryo and investigated their formation. They have also discussed the diagnosis and treatment of such urinary obstructions with the aid of cystoscopy and other modern surgical methods.

Congenital anomalies of the genito-urinary tract are found frequently in infants. Congenital cysts, however, are unusual and a case with its autopsy findings is herewith reported.

CASE REPORT

G. D. P., a white male infant, was born on August 19, 1940. The family history was without significance. There was no record of any abnormalities. The child weighed eleven pounds at birth and was said to have eaten well and thrived until he was seventeen days old. His mother reported that he had voided larger quantities of urine than did her first baby, but not more frequently.

On the seventeenth day the infant became cross, developed a fever, and appeared to have difficulty in urinating. A purulent discharge was seen issuing from the meatus. A physician was then called who found numerous white cells in a urinalysis and made a diagnosis of pyelitis. At this time the child did not seem to be particularly ill. For the next four days he was observed at home and given sulfanilamide. The temperature of the patient ranged from 101 to 103 F., and he voided regularly.

Four days after the onset of symptoms the child became definitely worse and was admitted to the University Hospital.

On admission the patient was cyanotic, apparently from the sulfanilamide which he had

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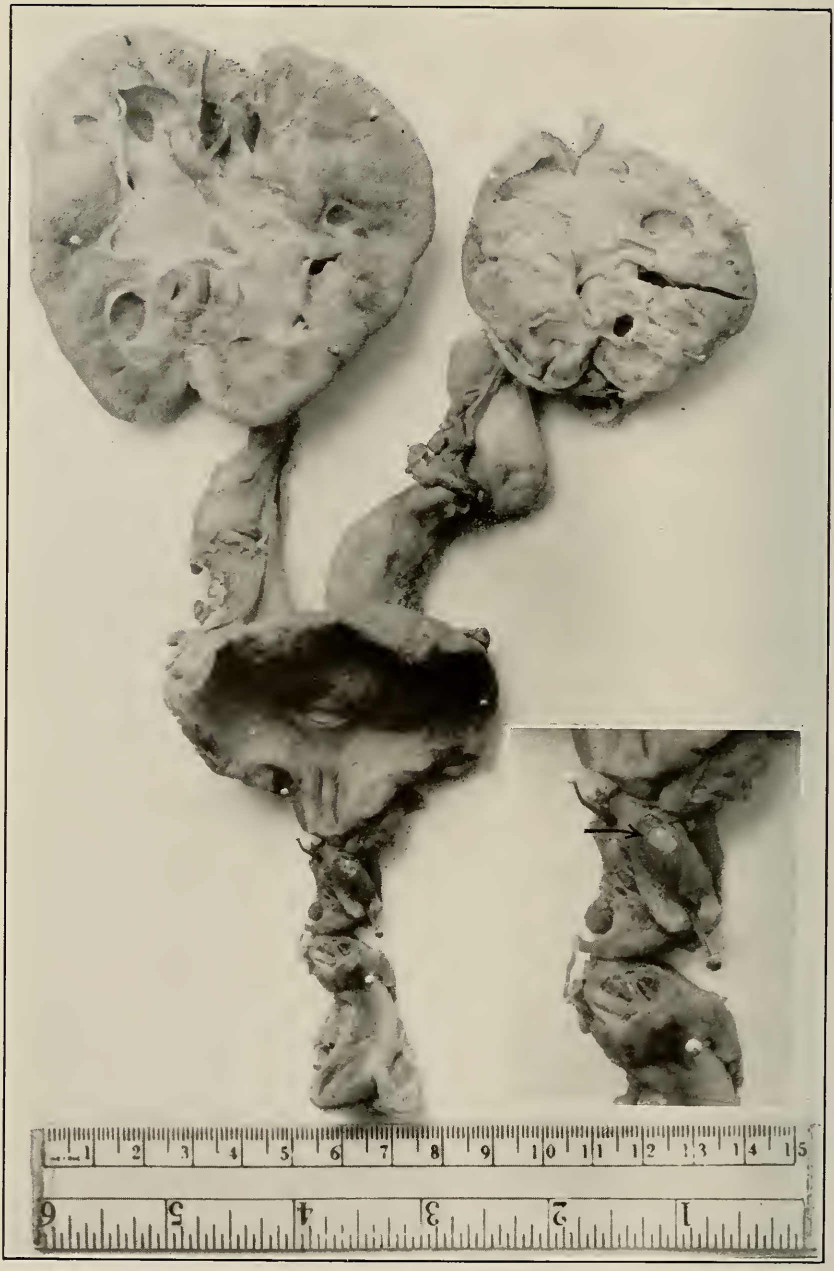

FIG. 1. A photograph of the genito-urinary tract as removed at autopsy. The inset is an enlargement of the cystic area.

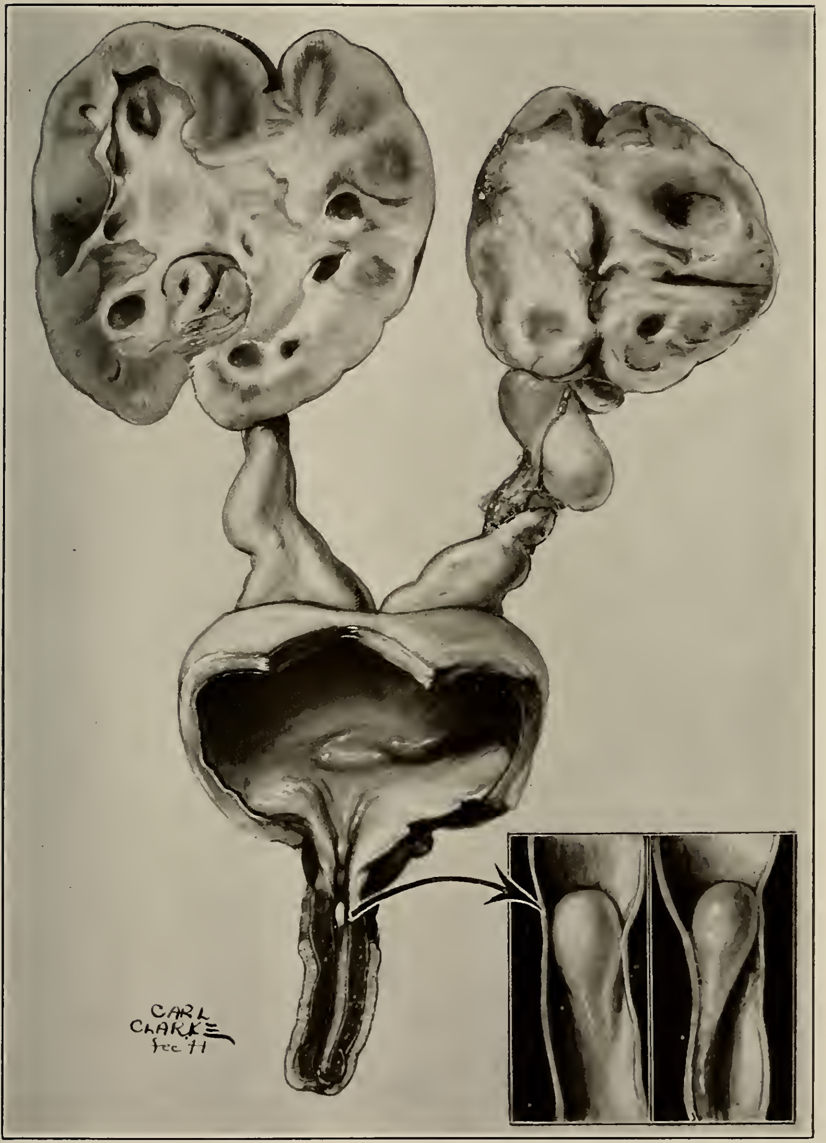

FIG. 2. A drawing showing the reconstructed genito-urinary tract. The inset shows the ball type of obstruction anchored to the wall of the urethra.

been given. He was toxic, restless, and obviously uncomfortable. The abdomen was distended and tympanic, and the baby vomited repeatedly.

Because of the child's obvious discomfort and respiratory difficulty the first effort was devoted to the alleviation of the abdominal distention. Enemas, the use of rectal tubes, and stomach lavage were ineffectual. After a few hours it was noted that he had passed no urine and that there was an area of dullness well above the pelvic brim. His small meatus prevented the use of ordinary catheters; however, a filiform sound was introduced without difficulty. Finally, a urethral catheter was passed into the bladder without meeting any apparent obstruction. About two ounces of thick, turbid urine were drained off and examination revealed this to contain innumerable white blood cells. A few hours later the child was again catheterized and more turbid urine obtained. However, his condition did not improve. He refused to eat, and abdominal distention and distress persisted.

In spite of transfusions and other supportive measures he became rapidly worse and died early the second day after admission to the hospital, which was the twenty-third day of life. Owing to the critical condition of the patient from the time of his admission, valuable and interesting studies which might otherwise have been made were not attempted.

AUTOPSY REPORT

The external examination revealed the body of a well-developed, well-nourished, white male infant. The abdomen, particularly below the umbilicus, was distended. The scrotum was slightly edematous.

An examination of the thoracic cavity disclosed that the heart was enlarged and weighed 45 grams, as compared to the average weight of the heart of a three weeks old child, which is 20 grams. This enlargement consisted of hypertrophy of the left ventricular wall and dilatation of the left ventricular cavity.

On excising the abdomen, the stomach and intestines were found to be distended with gas. The fundus of the urinary bladder reached to the umbilicus and it was evident upon palpation that the bladder was filled with fluid. Pressure exerted upon the bladder did not cause the passage of urine through the urethra. However, a sound was passed through the external urethral meatus and into the bladder without meeting an obstruction. The ureters were considerably dilated and tortuous. The kidneys could be palpated through the posterior peritoneum and conveyed the impression of being enlarged and cystic.

The distended bladder, when opened, was found to contain turbid urine which was grossly purulent. The wall of the bladder was definitely thickened and the mucous membrane was dull and roughened. Both ureteral openings into the bladder were enlarged. The ureters were dilated and tortuous, with a diameter in some areas of 15 to 18 mm.

The urethra was incised and examined. A small cystic oval mass was seen to be occluding the first portion of the membranous urethra distal to the prostatic portion. This cyst measured 2 mm. in diameter and was attached to the posterior wall of the urethra. The free surface was directed proximally up into the urethral lumen. This created a ball valve type of obstruction to the passage of urine from the bladder down through the urethra. The cystic mass occupied the anatomic position of the entrance of the ducts of the bulbo-urethral or Cowper's glands into the urethra.

The kidneys were abnormal in appearance. The capsules stripped with only slight difficulty and disclosed a slightly granular surface. The right kidney was enlarged and measured 8 by 5 by 3 cc. On palpation it was found to be soft and cystic. The left kidney was also enlarged and measured 4 by 3.5 by 2 cm. It was soft and even more cystic than the right kidney. Cut surfaces of both kidneys showed dilated pelves and calices, and roughened and dull mucous membranes. The previously mentioned cystlike appearance of the kidneys was caused by dilatation of the pelves and calices. There were no true cysts present. The cortex and medulla of each kidney were thin. The entire thickness of the cortex and medulla on the right was 8 mm., whereas the thickness on the left was 3 mm. Therefore, each kidney was made up of a small rim of remaining parenchymatous tissue surrounding the dilated calices, which were lined with a roughened dull membrane. There was more residual parenchymatous tissue substance in the right kidney than in the left. An examination of the cut surface of the distorted and scarred cortex and medulla revealed numerous small, pinpoint white areas which were probably abscesses.

Microscopic examination. The normal microscopic structure of each kidney parenchyma was considerably distorted by an extensive infiltration of inflammatory cells consisting of polymorphonuclear neutrophils, lymphocytes, and large macrophages. The polymorphonuclear neutrophil was the predominating cell. A collection of lymphocytes surrounded the vessel walls.

Many of the capsular spaces and tubules were filled with polymorphonuclear neutrophils. The interstitial tissue of both the cortex and the medulla was heavily infiltrated by these acute inflammatory cells. There were large conglomerate areas of acute inflammatory cells which formed abscesses in the cortex of the kidneys. The mucous membranes of the pelves and calices of the kidneys were replaced by a thick inflammatory layer of organizing granulation tissue. The tubular epithelium was in varying stages of degeneration. Some of the collecting tubules were dilated and contained clumps of acute inflammatory cells.

The left kidney, in addition, was involved by an irregular fibrosis, which in some areas extended from the cortex through the medulla to the pelvis. This tissue was severely compressed and scarred.

The mucous membrane of the bladder was replaced by an inflammatory layer of organizing granulation tissue.

The urethra immediately proximal to the cystic area showed dilated grandular structures lined with pseudostratified columnar epithelium, which were Cowper's ducts. These were directly beneath the urethral mucous membranes.

The small piece of urethra and the adjoining cyst were misplaced after the specimen had been photographed and therefore no section was available.

Final anatomic diagnosis. Congenital cyst of membranous urethra (probably arising from Cowper's glands); hypertrophy of the bladder wall; hydro-ureter (bilateral); hydronephrosis (bilateral); cystitis, pyelonephritis (bilateral); pyonephrosis (bilateral); cardiac hypertrophy, pulmonary congestion, and emphysema of the lower lobes (bilateral).

COMMENT

Apparently the cystic mass which blocked the urethra represented the result of a congenital closure of the duct of one of Cowper's glands and caused an accumulation of secretion in the gland. Since this gland lies just beneath the mucous membrane of the urethra, the mass protruded into the urethra and created an obstruction to the flow of urine.

During the development of the fetus the bulbomembranous, or Cowper's glands, grow from the membranous urethra as epithelial rests, branch, acquire a lumen lined with secretory epithelium, and open into the deep bulbous urethra.

In addition to the normal glands there sometimes develop in the bulbous urethra accessory Cowper's glands, the ducts of which open into the urethra proximal to those of the main ducts. These accessory glands are short and lie nearer to the urethra than the main ducts but parallel to them. Any of these ducts can, of course, be shut off or be congenitally absent, so that the secreting gland forms a cyst which may block the lumen of the urethra.

In an embryo measuring 75 mm., which is the average size at the end of the third month of intra-uterine life, the ducts of Cowper's glands show definite lumen and the beginning of branches. Following the conversion of the ramified branches into alveoli lined with mucus secreting cells, Cowper's glands begin to secrete when the embryo reaches about 120 mm. in size, or at the end of the fourth month of intra-uterine life. The kidneys begin to secrete at about the third month of intra-uterine life. Hence, a cyst in the urethra may form early in intra-uterine life. Such a cyst can and sometimes does block the passage of urine, with severe or irreparable damage to the urinary organs long before birth.

SUMMARY

A case of a congenital cyst, apparently arising from a Cowper's gland in the membranous urethra of a three weeks old male infant is reported. The urinary blocking caused by this cyst was responsible for a severe hydronephosis with destruction of most of the kidney tissue, dilated ureters, bladder wall hypertrophy, cardiac hypertrophy, and other pathologic changes in the body. Owing to the extent and severity of the anatomic changes noted in the genito-urinary tract, especially in the kidneys, a condition of partial urethral block must have existed for a considerable period of intra-uterine life.

It is noteworthy, however, that until a widespread infection of the urinary tract occurred approximately two weeks after birth there was no clinical evidence that the child was abnormal.

Since inflammatory changes of the genito-urinary tract frequently result from congenital abnormalities, the presence of evidence of such inflammation should always lead to a thorough investigation of the possibility of congenital defects of the genito-urinary tract.

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SODIUM TETRADECYL SULFATE USED IN THE TREATMENT OF ACUTE PYOGENIC EMPYEMA OF THE PLEURAL CAVITY*†

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The search for a solvent of sputum and empyema fluid for concentration examination for tubercle bacilli led Dr. S. A. Petroff‡ to the use of sodium tetradecyl sulfate. This product definitely has found its place in the commercial field as a detergent and wetting agent (1, 2).

Sodium tetradecyl sulfate, called *Tergitol*, was found to have a bactericidal as well as a solvent action. It was noted that there was an enhancement of the bactericidal properties of well-known antiseptics by the addition of Tergitol (3).

Tergitol-azochloramide solution has been used in the treatment of pure tuberculous empyemas and in mixed infection tuberculous empyemas with success (4). Its use in the treatment of pneumococcal and streptococcal empyemas has been mentioned (3).

The authors have used Tergitol solutions in the treatment of tuberculous empyemas successfully and have had beneficial results with the solution in chronic empyemas and infected wounds. Two acute postpneumonic empyemas were treated and remarkable results followed. Since the outcome in each instance was so outstanding, these two cases are being reported.

CASE REPORTS

Case No. 1: D. R., Church Home and Infirmary, No. 25,226, was a white woman, age 27 years. She was admitted on April 21, 1941 and discharged on May 19, 1941.

In February, 1941 the patient had pneumonia and three weeks later yellow sterile fluid was aspirated from the left pleural cavity. She had been discharged from the hospital, but continued to have fever and reported that she never felt well. Four days before admission she began coughing up considerable puslike material. The past and family histories were noncontributory.

Physical examination revealed an obese, obviously ill, white woman. There were typical findings of hydropneumothorax on the left side of the chest (Fig. 2). The abdomen presented a large postoperative hernia. The findings of routine laboratory tests were negative.

A roentgenograph of the chest revealed a large collection of fluid and air in the left thoracic cavity (Fig. 2). The right lung was normal. Aspiration of the pleural cavity revealed thick, yellowish-green, foul-smelling pus. A smear of the pus showed many

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* From the Department of Surgery, School of Medicine, University of Maryland.

† Received for publication October 10, 1941.

‡ The sodium tetradecyl sulfate was kindly furnished by Dr. S. A. Petroff.

gram-positive and short chain cocci. There were no tubercle bacilli. The culture of the pus showed no growth of bacteria.

Thoracentesis was done on April 22, 1941, and on each successive day for three days, with no change in temperature. At the third aspiration methylene blue was instilled into the pleural cavity and the patient coughed it up, thus proving the presence of a bronchopleural fistula.

April 25, 1941: Treatment with azochloramide 1:3300 in Tergitol 1:500 was started. There was aspirated from the pleural cavity 150 cc. of thick, foul-smelling, methylene blue-stained fluid; 100 cc. of Tergitol-azochloramide solution was instilled. The patient coughed up some of this instilled fluid. Within twelve hours the temperature dropped to normal (Fig. 1).

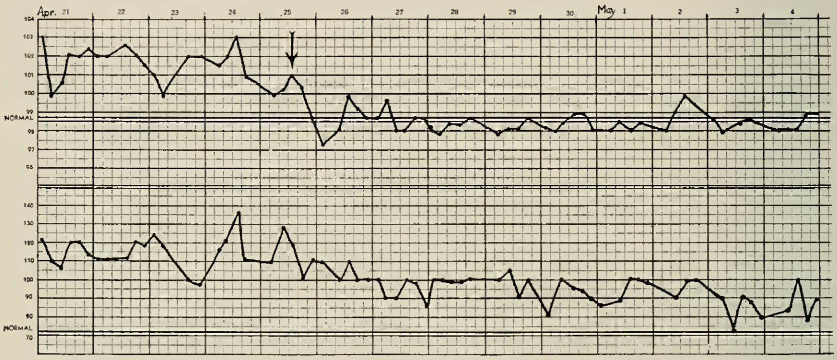


FIG. 1. A photograph of a portion of the temperature chart of D. R. It shows prompt response to treatment; the arrow indicates the time of first aspiration of the pleural cavity and instillation of Tergitol-azochloramide solution. The remainder of the chart reveals normal temperature.

April 26, 1941: The pleural cavity was aspirated and the fluid obtained appeared much like that of the day before but had no odor (Fig. 5); 100 cc. of Tergitol-azochloramide solution was instilled. The aspirated fluid was cultured but there was no growth.

April 27, 1941: A thoracentesis was done and thinner fluid was obtained; 100 cc. of Tergitol-azochloramide was instilled. There was no coughing.

April 28, 1941: The pleural cavity was aspirated, 100 cc. of fairly clear thin fluid being obtained; 100 cc. of Tergitol-azochloramide solution was instilled. Severe coughing followed. The patient was placed on the left side and the cough subsided. She continued free of symptoms and no aspiration was done until four days later.

May 2, 1941: Aspiration of the chest revealed only 40 cc. of fluid. A negative pleural pressure was obtained, which indicated closure of the bronchopleural fistula. Sixty cc. of Tergitol-azochloramide solution was instilled. There was no coughing.

May 7, 1941: A thoracentesis yielded 30 cc. of fluid; 60 cc. of Tergitol-azochloramide solution was instilled. No growth of bacteria was shown in a culture of the aspirated fluid.

May 12, 1941: A thoracentesis revealed 40 cc. of fluid; 40 cc. of Tergitol-azochloramide solution was instilled (Fig. 3).

May 19, 1941: Aspiration of the pleural cavity produced 25 cc. of fluid similar to that injected one week before; 25 cc. of Tergitol-azochloramide solution was instilled.

The patient was discharged from the hospital after having had eight aspirations of the chest, with instillations of Tergitol-azochloramide solution. After the second day of treatment the temperature was constantly normal, except for one day when it was 100 F.

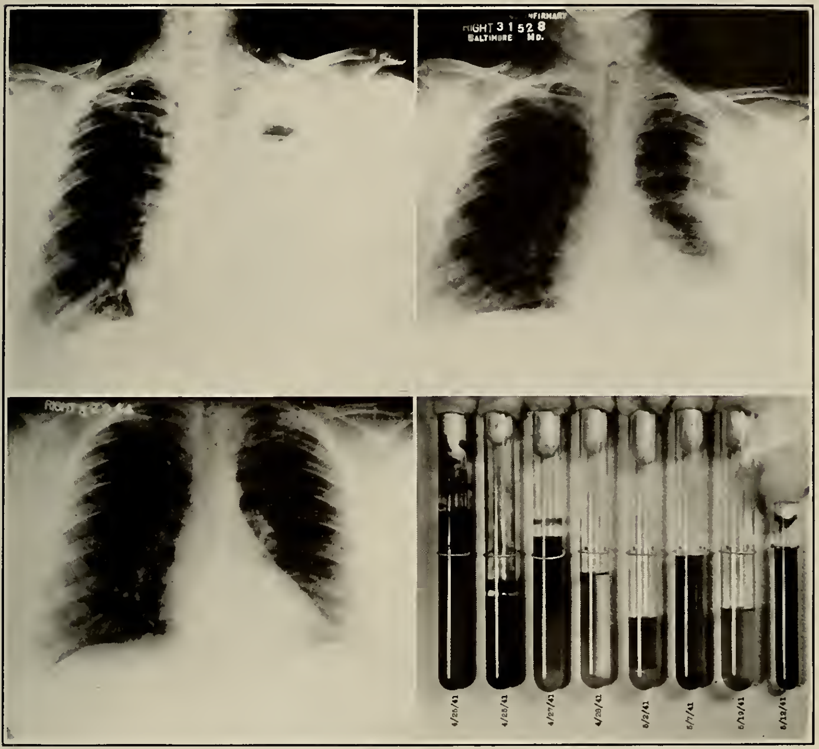


Fig. 2. (Upper left) Photograph of roentgen-ray film (D. R.) taken April 22, 1941, before treatment of empyema was begun.

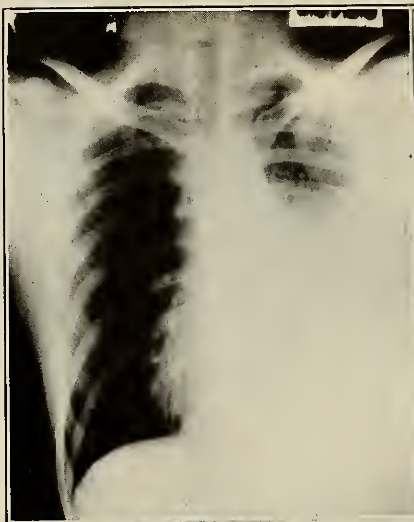
Fig. 3. (Upper right) Photograph of roentgen-ray film (D. R.) taken May 13, 1941, twenty-one days after treatment of empyema was begun.

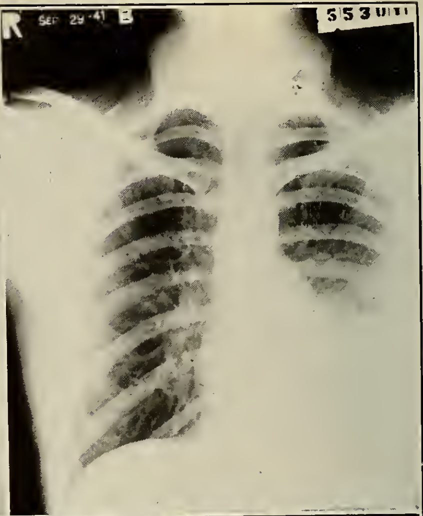
Fig. 4. (Lower left) Photograph of roentgen-ray film (D. R.) taken July 29, 1941, three months after beginning treatment of empyema and approximately two months after all treatment was discontinued. It shows the end result.

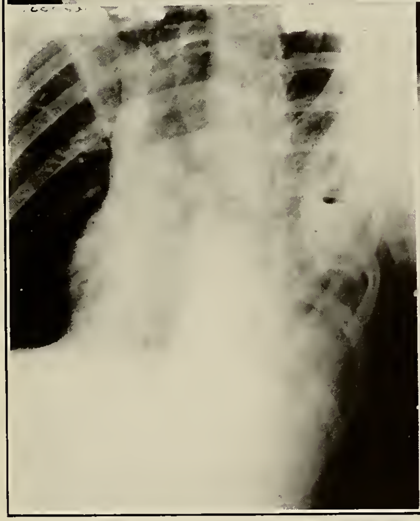
Fig. 5. (Lower right) Photograph of the material aspirated from the pleural cavity of D. R. It shows clearly the changing character of the pleural fluid as treatment progressed satisfactorily. The first three tubes contain methylene blue which had been instilled before treatment was begun. It emphatically indicates the inability to aspirate all the pleural fluid at any one aspiration.

by mouth. Her clinical condition was asymptomatic. During the hospital stay sulfathiazole was given by mouth.

After being discharged from the hospital the patient was followed carefully. Her own temperature chart showed no fever for a period of six weeks.







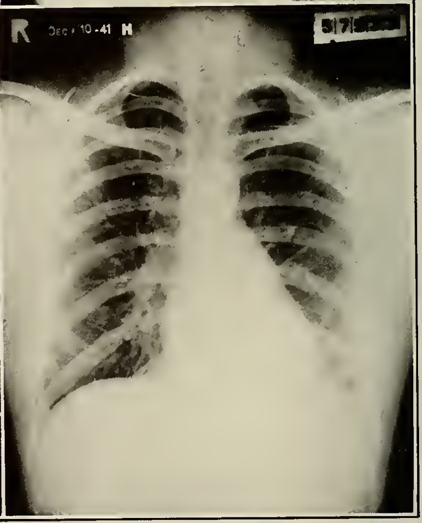


FIG. 6. (Upper left) Photograph of roentgen-ray film (H. K.) taken September 9, 1941, before treatment of empyema was begun.

FIG. 7. (Upper right) Photograph of roentgen-ray film (H. K.) seventeen days after treatment of empyema was begun.

FIG. 8. (Lower left) Photograph of roentgen-ray film (H. K.) taken seventeen days after treatment of empyema was begun. When compared with Fig. 7 the added information obtained by films other than the usual anterio-posterior view is noted.

FIG. 9. (Lower right) Photograph of roentgen-ray film (H. K.) taken December 10, 1941, three months after beginning treatment of empyema and approximately two months after all treatment was discontinued. It shows the end result.

June 2, 1941: The pleural cavity was aspirated and only a few cubic centimeters of fluid were obtained; no instillation was done. A culture of this fluid showed no growth of bacteria.

July 29, 1941: A roentgenograph of the chest showed that the left side of the diaphragm was slightly elevated and there was a slight thickening of the pleura (Fig. 4). The patient was feeling well and had no complaints.

Case No. 2: H. K., a white male, age 24 years, was admitted to the University Hospital on August 23, 1941, and discharged on October 4, 1941.

The illness began one week before admission, with pain in the left side of the chest, cough with brownish expectorations, weakness and exhaustion. The patient was examined by Dr. J. E. Muse, Jr., who made a diagnosis of pneumonia and advised admission to the hospital. Past and family histories were noncontributory.

Physical examination revealed a well-nourished, ill-looking patient. The chest findings were typical of pneumonia in the left lower lobe. The remainder of the physical examination was normal.

Roentgenographic examination of the chest confirmed the findings of pneumonia in the left lower lobe. The patient was given adequate chemotherapy, with slow recovery. After a short period, which was free of fever, he again began to have an elevation of temperature. The development of empyema was suspected.

September 9, 1941: A roentgenograph of the chest showed a considerable quantity of fluid in the left pleural cavity, which displaced the heart and mediastinum to the right (Fig. 6).

September 10, 1941: The pleural cavity was aspirated. Culture of this fluid revealed staphylococcus albus, streptococcus hemolyticus, and a gram-negative rod, with a nonspecific reaction on differential media.

September 12, 1941: Treatment with azochloramide 1:3300 in Tergitol 1:500 was started. The pleural cavity was aspirated of 400 cc. of thick odorless fluid which seemed typical of pneumococcic pus; 100 cc. of Tergitol-azochloramide solution was then instilled. The temperature was 103 F. by mouth.

September 13, 1941: The pleural cavity was aspirated of 175 cc. of fluid that was thinner than on the preceding day; 150 cc. of Tergitol-azochloramide solution was instilled. The patient coughed at the termination of treatment but did not expectorate any of the instilled fluid. The temperature was 99 F. by mouth; the patient looked and felt well as compared with his condition on the preceding day.

September 14, 1941: A total of 160 cc. of thin fluid was aspirated from the pleural cavity; 170 cc. of Tergitol-azochloramide solution was instilled. The temperature was normal and for this reason aspiration was not done the next day.

September 16, 1941: The pleural cavity was aspirated of 185 cc. of thin fluid; 160 cc. of Tergitol-azochloramide was instilled. Culture of the aspirated fluid showed no growth of bacteria in forty-eight hours.

September 17, 1941: Aspiration of the pleural cavity revealed 150 cc. of fluid; 130 cc. of Tergitol-azochloramide solution was instilled.

September 19, 1941: The pleural cavity was aspirated of 100 cc. of fluid; 40 cc. of Tergitol-azochloramide solution was instilled. The patient coughed up a small amount of the instilled fluid. He was placed on the affected side and the cough stopped. The symptoms definitely suggested a bronchopleural fistula. However, he had not coughed since the treatment on September 13, 1941.

September 21, 1941: Thirty cc. of fluid was aspirated from the pleural cavity; 90 cc.

of Tergitol-azochloramide solution was instilled. Again he coughed up some of the instilled fluid.

September 24, 1941: Thirty cc. of fluid was aspirated from the pleural cavity. A definite negative pressure was obtained after aspiration, which indicated that the bronchopleural fistula had closed. There was instilled 90 cc. of Tergitol-azochloramide solution. The patient did not cough during or after this treatment.

September 29, 1941: A pleural aspiration revealed only 40 cc. of thin fluid, or an absorption of 50 cc. since the previous instillation; 40 cc. of Tergitol-azochloramide solution was then instilled. A roentgenograph of the chest revealed considerable clearing since the last film was taken (Fig. 7). There was a small encapsulated area of hydropneumothorax (Fig. 8).

The patient's temperature continued normal after September 15, 1941, at which time it was 99 F. by mouth. He received sulfathiazole by mouth during this treatment. The patient was discharged from the hospital on October 4, 1941.

October 6, 1941: A pleural aspiration revealed 40 cc. of fluid; 30 cc. of Tergitol-azochloramide was then instilled.

October 17, 1941: The pleural cavity was aspirated and only 15 cc. of fluid was obtained; 10 cc. of Tergitol-azochloramide was instilled.

October 27, 1941: The patient had no symptoms and felt well. All physical findings were normal, except for a lag on the left side of the chest.

The procedure recommended for the treatment of acute pyogenic empyemas of the pleural cavity with Tergitol is based on experience obtained from its use in tuberculous, chronic pyogenic, and acute pyogenic empyemas. The aspirating needle should be of large enough bore (16 or 18) to allow free aspiration. It should be fitted with a needle guard in order to prevent the needle from entering too deeply into the chest during treatment. If the needle moves out of the chest wall somewhat it can cause no harm, unless fluid is injected inadvertently. When a needle guard is used under such circumstances, the needle can be reinserted to the proper depth. After aspiration, the patient should be placed with the site of the needle puncture uppermost, to prevent a leakage of the fluid into the tissues. Absolute asepsis should be maintained. A negative intrapleural pressure should be obtained at the end of each treatment in order to hasten rapid reexpansion of the lung and pleural synthesis. As the pleural space is obliterated the residual pleural cavity must be followed carefully. In order to find the remaining pleural space it may be necessary to check the location by roentgenographic examination. The solution found most satisfactory has been azochloramide 1:3300 in Tergitol 1:500. The azochloramide may be used in a strength of 1:1600. The quantity of Tergitol solution injected at the first treatment should not exceed 100 cc. This amount may be increased up to 300 cc. in subsequent injections, depending upon the response of the patient and the size of the pleural space. Likewise, as the pleural space is obliterated, the quantity must be decreased in order to maintain a negative pleural pressure. The pleural cavity should be aspirated and instilled on four successive days at the beginning of treatment. The interval of aspiration and instillation can then be lengthened, according to the response of the patient and the reports of bacterial cultures. A bacterial culture of the pleural fluid should be made at each aspiration. If satisfactory response is not obtained in four to six days, treatment should be discontinued in favor of one of the well-established, time-tested methods for treatment of acute empyema.

The action of the Tergitol solution is not specific and therefore promises a wide scope of usefulness. Further study of this subject is being made.

CONCLUSIONS

Two cases of acute pyogenic empyema of the pleural cavity have been cured by aspiration of the pleural cavity and instillation of Tergitol-azochloramide solution. The prompt response to treatment and the shortened convalescence make this method of treatment worthy of further investigation.

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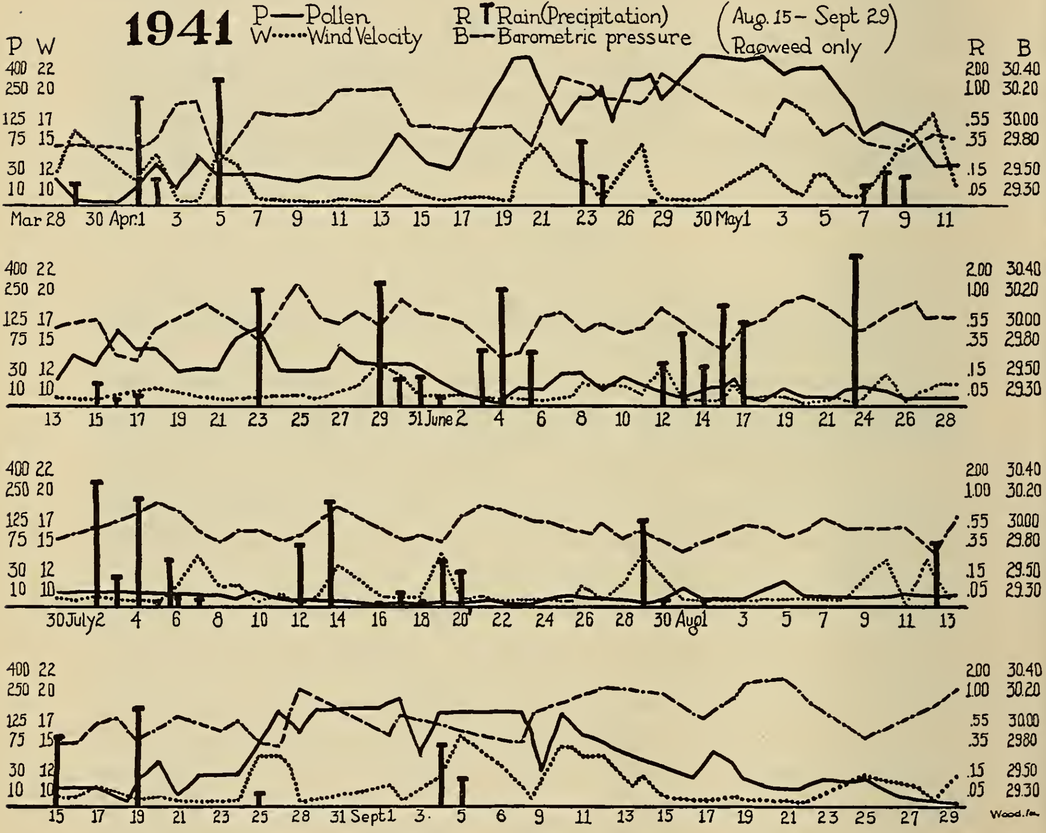
POLLEN COUNTS*†‡

HOWARD M. BUBERT, M.D. AND JANE A. WOOD, A.B.

BALTIMORE, MD.

As for the past four years, the Allergy Clinic presents herewith the results of the pollen counts made for the Baltimore area from March 28 to September 29, 1941.



COMMENT

It is interesting to note that in contrast to the previous year the spring pollen began around the middle of April and the count continued high until the second week in May. The ragweed pollen appeared late again but did not reach an equal concentration with that of 1940.

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* From the Allergy Clinic, Department of Medicine, School of Medicine, University of Maryland.

† Received for publication February 21, 1942.

‡ Pollen counts supervised by Jane A. Wood.

METHOD

The method used was the same as described in 1939 and will not be repeated here. Previous counts and a description of the method employed have been reported for these four years (1, 2, 3, 4).

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(2) Bubert, H. M. and Rosenberg, S.: Pollen Counts. Bull. School of Med., Univ. of Med., **23**: 179, 1939.
(3) Bubert, H. M. and Rosenberg, S.: Pollen Counts. Bull. School of Med., Univ. of Md., **24**: 239, 1940.
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PROCEEDINGS
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ONE HUNDRED AND SIXTEENTH PROGRAM MEETING

The One Hundred and Sixteenth Program Meeting was held on Wednesday, December 10, 1941, at 4:30 p.m. in the Bressler Lecture Hall. The following program was presented:

"A New Method for Preserving Hemoglobin without Methemoglobin Formation," by Miss Joye E. Jacobs, Department of Physiology, School of Medicine, University of Maryland.

"Animal Tests with Human Hemoglobin Solutions as a Blood Substitute," by Dr. William R. Amberson, Department of Physiology, School of Medicine, University of Maryland.

"Sixteen Millimeter Color Film of Autopsy Technic," by Dr. Henry Wollenweber, Departments of Pharmacology and Pathology, School of Medicine, University of Maryland.

Abstracts of these papers follow.

A NEW METHOD FOR PRESERVING HEMOGLOBIN WITHOUT METHEMOGLOBIN FORMATION

Joye E. Jacobs, A.B.*

Human hemoglobin solutions made isotonic with blood plasma are sterilized by Seitz filtration. The solutions are completely reduced by vacuum extraction with a Hyvac pump. They are then sealed in vacuo in glass ampules. Although exposed to light and warm room temperatures these solutions show no methemoglobin formation for periods up to eight months.

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* From the Department of Physiology, School of Medicine, University of Maryland.

ANIMAL TESTS WITH HUMAN HEMOGLOBIN SOLUTIONS AS A BLOOD SUBSTITUTE

William R. Amberson, Ph.D.*

Sterile evacuated solutions of human hemoglobin, properly balanced and buffered, and kept without refrigeration for as long as eight months, have been transfused into cats with the simultaneous removal of equal amounts of normal blood. The animals survive after replacement of as much as 85 per cent of normal blood, without pyrogenic reaction or asphyxial signs. Blood pressures tend to be above normal in the first hours after replacement. Hemoglobinemia and hemoglobinuria terminate in from thirty-six to forty-eight hours. No tissue pathology has been noted, but hyperemia of the liver usually persists for several weeks, and cells of the reticulo-endothelial system show hemoglobin inclusions for some time.

ONE HUNDRED AND SEVENTEENTH PROGRAM MEETING

The One Hundred and Seventeenth Program Meeting was held on Tuesday, January 20, 1942, at 4:30 p.m. in the Bressler Lecture Hall. The following paper was presented:

"The Resurrectionists," by Dr. Marion W. McCrea, Departments of Histology and Embryology, School of Dentistry, University of Maryland.

An abstract of this paper follows.

THE RESURRECTIONISTS

Marion W. McCrea†

The early methods for obtaining bodies for anatomic dissection were described, together with the improvements in the teaching of anatomy beginning with the Galenic period. A discussion pertaining to the influence of Vesalius on anatomic study was given. This was followed by a brief account of the development of the medical and anatomic schools in England, Scotland, Ireland and Wales during the period when body snatchers and resurrectionists reached their peaks. Illustrative cases were cited to demonstrate the methods employed by these individuals who were dealing in "scientific goods." Examples were given which showed the means used to combat the ravages of the resurrectionists. Finally, consideration was given to the history of the Warburton Act that was passed by Parliament in 1832. This act legalized the supply of dissection material for anatomy. The background of British experience was paralleled, though on a smaller scale, during the early stages of anatomic study in this country. Most of the legislative measures relating to the teaching of anatomy passed by the various states showed marked similarity to the provisions of the Warburton Act.

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* From the Department of Physiology, School of Medicine, University of Maryland.

† From the Departments of Histology and Embryology, School of Dentistry, University of Maryland.

ONE HUNDRED AND EIGHTEENTH PROGRAM MEETING

The One Hundred and Eighteenth Program Meeting was held on Wednesday, February 25, 1942, at 4:30 p.m. in the Bressler Lecture Hall. The program consisted of the following papers:

"The Effect of Adrenalin on the Oxygen Consumption of the Fish, Girella Nigricans," by Drs. Dietrich C. Smith and Samuel A. Matthews, Department of Physiology, School of Medicine, University of Maryland, and Department of Biology, Williams College, Williamstown, Massachusetts.

"The Innervation of the Thyroid Gland," by Dr. Harry A. Teitelbaum, Department of Gross Anatomy, School of Medicine, University of Maryland.

Abstracts of these papers follow.

THE EFFECT OF ADRENALIN ON THE OXYGEN CONSUMPTION OF THE FISH, GIRELLA NIGRICANS

DIETRICH C. SMITH, Ph.D.* AND SAMUEL A. MATTHEWS, Ph.D.†

Adrenalin in doses ranging from 0.5 to 0.2 cc. of 1:1000 dilution depresses the oxygen consumption of the fish, Girella nigricans. This fall in oxygen utilization is always accompanied by a paling of the animal. The larger doses usually lead to death of the fish in one to two hours, the oxygen consumption sinking progressively during this time. With smaller doses the fall is on the average to about one-half the preinjection level and is maximal in thirty minutes, recovery being complete in about sixty to ninety minutes. Ineffective doses, as a rule less than 0.5 cc. of 1:10,000 dilution, are not accompanied by any color change, thereby indicating that this dose is also below the threshold capable of producing concentration of the melanophore pigment. The effect on oxygen consumption of such doses is exactly the same as that of control injections of 0.5 to 1 cc. of sea water or 0.3 cc. of 0.5 per cent chloretone, namely, a rise in oxygen consumption of about 50 per cent, maximal in one hour, followed by a return to normal within two to three hours. This rise is attributed to the effects of handling the fish rather than to any specific effect of the injected substance.

THE NERVE SUPPLY TO THE THYROID GLAND OF TRITURUS TOROSUS

HARRY A. TEITELBAUM, M.D., Ph.D.‡

The thyroid gland of the salamander, Triturus torosus, was stained with methylene blue. The animal was perfused with the dye by means of a cannula inserted into the ventricle and tied in the aortic conus. Following fixation and clearing, the whole unsectioned thyroid was mounted in balsam and studied. The observations reported are preliminary and are still being consulted for purposes of interpretation.

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* From the Department of Physiology, School of Medicine, University of Maryland.

† From the Department of Biology, Williams College, Williamstown, Mass.

‡ From the Department of Gross Anatomy, School of Medicine, University of Maryland. This work was carried out with the aid of the Weaver Fellowship Fund of the School of Medicine, University of Maryland.

The hypoglossal nerve, which lies in contact with the thyroid, was not found to give any fibers to that gland. The nerves that entered the latter did so in company with blood vessels. Some individual fibers could be traced through large parts of the gland under lower magnifications.

The nerve fibers observed were either isolated or in bundles. An occasional myelinated fiber was seen. In addition, extensive networks of fibrous tissue were present throughout the thyroid and ramified over the follicles and in the interfollicular spaces. Some of these fibers formed layers which took curved courses, as though conforming with the contour of a follicle. Others formed irregular spirals and might be related to blood vessels.

The finer ramifications of the fibers stained with methylene blue could be seen on the surfaces of the thyroid cells. Small varicosities and ring structures lay in contact with the cells. There were also fine, wavy, intercellular fibers that presented a particularly difficult problem of interpretation.

Recent observations of Nonidez (1935) cast doubt on the nature of a considerable amount of the stainable fibrous tissue in the thyroid. He expressed the opinion that much of this that has been interpreted as nervous by Boeke and Sunder-Plassman was really connective tissue. The findings reported herein are subject to the same problems of interpretation as are the observations of all other investigators. Only by means of careful study with highly differential staining technics will a solution be reached.

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SPRING ACTIVITIES

The Board of Regents, the President and the Board of Directors of the Alumni Association, together with the Acting Dean and Medical Council of the School of Medicine of the University of Maryland, cordially invite you to attend the spring activities.

PROGRAM

May 28, 1942

9:00 A.M.—Registration at the Students' Lounge, first floor, Gray Laboratory.

10:00 A.M.—12 M.—Inspection of University Hospital and School of Medicine.

1:00 P.M.—Luncheon and Annual Meeting of the Medical Alumni Association, Nurses' Dining Room, University Hospital.

7:00 P.M.—Annual Banquet, Lord Baltimore Hotel.

REUNIONS

The following classes have expressed their intention to hold reunions this year:

1892
1897
1902
1907
1912
1917
1922
1927
1932
1937

NOTE: The reunions will include the graduates from the College of Physicians and Surgeons, University of Maryland and the Baltimore Medical College. Any other classes desiring to hold reunions will please communicate with the Medical Alumni Office, Lombard and Greene Streets, Baltimore, Maryland, which will be pleased to help in every possible way to make the affair a success.

MAY 30, 1942

11:00 A.M.—Commencement Exercises, Ritchie Coliseum, College Park, Md.

Secretary of the Medical Alumni Association.

Kindly send . . . . . . ticket . . for the Alumni Banquet of the Medical Alumni Association, University of Maryland, to be held at the Lord Baltimore Hotel on Thursday evening, May 28, 1942 at 7 P.M.

will
I *or* be present for the banquet.
will not

Enclosed please find subscription at $2.50 per ticket.
Name. . . . .
Address. . . . .

(*Detach and mail to:* MEDICAL ALUMNI ASSOCIATION
Lombard and Greene Sreets, Baltimore, Md.)

Programs may be obtained at the time of registration.

ITEMS

Lieut. Col. Clyde C. Johnston, (B.M.C. 1909) Medical Corps, Regular Army, has been assigned to the post of director of the Department of Extension Courses at the Medical Field Service School, Carlisle Barracks, Pa. Colonel Johnston has been in service at many posts within and without the United States since he was first commissioned in 1920.

Colonel Johnston is a graduate of Kearney Military Academy and the University of Pennsylvania, and also attended three Army Service Schools, the Medical Field Service School, the Army Medical School, and the School of Aviation Medicine. He came to Carlisle Barracks from Camp Bowie, Texas, where he was commanding officer of the 64th Medical Regiment.

Capt. Louis Teitel, Class of 1935, has been placed in charge of the eye, ear, nose and throat section of the hospital at the Air Corps Basic Training School near Enid, Okla.

Before receiving his degree in medicine Captain Teitel attended the

University of Maryland at College Park and was given a Bachelor of Science degree in 1931. He furthered his studies as a physician at the Medical Field Service School at Carlisle Barracks, Pa. and was stationed at Camp Lee, Va. from May through November, 1941.

MEDICAL LIBRARY NOTES

One of the main objectives of the Library is the acquisition and preservation of material written by the alumni and staff of the School of Medicine. It would help materially if members of the University would send two reprints of their articles or a copy of their books to the Library for permanent preservation. The reprints will be bound and titled "Contributions from the Alumni and Staff."

The Library wishes to express its sincere gratitude to those who have contributed to the collection during the past year. A complete list of authors and titles would be too long for inclusion at this time. However, it is hoped that this collective expression will reach out and find its individual destination.

EXHIBIT AT ENOCH PRATT FREE LIBRARY

As in 1941, the Enoch Pratt Free Library has scheduled an exhibit of publications written by members of the faculties of Goucher College, Johns Hopkins University, Loyola College, and the University of Maryland. Members of the faculty of the University of Maryland have already been asked to submit material for inclusion to the University librarian who is responsible for our participation. The exhibit will be held during the month of April and will undoubtedly attract much attention, if last year's experience is any criterion.

DR. JOHN CRAWFORD

An article by Miss Julia Wilson, of the Library staff, entitled "An Early Baltimore Physician and his Medical Library," appeared in the January, 1942 issue of the Annals of Medical History. It is an account of Dr. John Crawford's life and library and is of particular interest to the School of Medicine, since Dr. Crawford's collection of books, purchased upon his death in 1813, formed the nucleus of the Library.

Dr. Stuart D. Sunday, Class of 1936, has been promoted to a captaincy in the Army Medical Corps and is now medical director of Fort Read Hospital, Trinidad, British West Indies.

Dr. Louis H. Douglass, Professor of Obstetrics, delivered an address on "The Problems of the Posterior Occiput" at the meeting of the Maryland

Academy of Medicine and Surgery which was held on March 17, 1942. On the same program Dr. Irving J. Spear, Professor of Neurology, spoke on "Recent Advances in the Diagnosis and Treatment of Nervous and Mental Disorders."

At the meeting of the Maryland Academy of Medicine and Surgery held on January 20, 1942 a paper on "The Treatment of Liver Diseases" was read by Dr. Thomas P. Sprunt, Professor of Clinical Medicine.

OBITUARIES

CHARLES M. PETERS, M. D.

Dr. Charles M. Peters, P. & S. 1913, physician and dentist, who was president of the New Jersey State Board of Dental Examiners from 1932 to 1938, died on December 27, 1941 at his home in Jersey City, New Jersey.

Dr. Peters was born in Richmond, Virginia. He started practicing dentistry in 1910 after his graduation from New York University. During the World War he had charge of oral surgery in St. Mary's Hospital in Hoboken, which had been taken over by the Army. He was in charge of oral surgery at North Hudson Hospital in Weehawken and Christ Hospital in Jersey City. Dr. Peters was a consultant at the Medical Center in Jersey City.

Bowlus, Edward Lingan, Baltimore, Md.; class of 1906; aged 69; died, December 31, 1941, of arteriosclerosis.

Brown, John P., Fairmont, N. C.; class of 1883; aged 77; died, December 7, 1941, of uremia.

Campbell, Eldridge H., Alderson, W. Va.; P. & S., class of 1894; aged 73; died, December 21, 1941, of coronary thrombosis.

Daudelin, Alfred, Nashua, N. H.; B.M.C., class of 1908; aged 63; died, September 13, 1941, of cardiorenal disease.

Dwyer, James Eugene, Milwaukee, Wis.; class of 1905; served during the World War; died, November 14, 1941, of nephrosclerosis.

Enlind, K. Arvid, New York City; P. & S., class of 1893; served during the World War; aged 68; died, November 17, 1941, in the Veterans Administration Facility.

Grady, William Earle, Tryon, N. C.; class of 1894; aged 70; died, December 9, 1941, of coronary thrombosis.

Leonard, John Michael, Fall River, Mass.; P. & S., class of 1900; aged 65; died, October 26, 1941, of carcinoma of the rectum.

Marsh, William Henry, Solomons, Md.; class of 1876; aged 90; died, November 12, 1941, in the United States Marine Hospital, Baltimore, of carcinoma of the lung.

Mathews, William, Philadelphia, Pa.; class of 1897; aged 76; died, November 1, 1941, of arteriosclerosis.

McCuskey, Laverty H., Moundsville, W. Va.; P. & S., class of 1897; aged 71; died, December 18, 1941, of coronary thrombosis.

Moe, Ray Wallace, Peekskill, N. Y.; P. & S., class of 1903; aged 61; died, October 27, 1941, of carcinoma of the lung and rheumatoid arthritis.

O'Neil, Terence Bernard, Ilion, N. Y.; B.M.C., class of 1901; aged 66; died, December 7, 1941, of heart disease.

Pate, Frederick James, Greensboro, N. C.; class of 1908; served during the World War; aged 56; died, December 14, 1941, of carcinomatosis.

Peters, Charles Manley, Jersey City, N. J.; P. & S., class of 1913; aged 62; died, December 26, 1941.

Quillen, Ralph Dwight, Baltimore, Md.; P. & S., class of 1909; aged 57; died, October 20, 1941, in a local hospital of arteriosclerosis and heart disease.

Simpers, Isaac Newton, Gaithersburg, Md.; B.M.C., class of 1891; aged 73; died, December 28, 1941, of heart disease and nephritis.

Suter, John Ellsworth, Piedmont, W. Va.; P. & S., class of 1893; aged 71; died, November 29, 1941, of chronic myocarditis.

Teter, J. M., Beaver, W. Va.; B.M.C., class of 1896; aged 68; died, December 10, 1941.

Thornton, David H., Princeton, W. Va.; P. & S., class of 1893; aged 76; died, November 19, 1941, in Roanoke, Va., of coronary sclerosis and hypertension.

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